This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
L41 .A421 4 STOR
A system of medicine, by many writers /
24503448325

LANE

MEDICAL

LIBRARY

LEVI COOPER LANE FUND

LIBRARY
OF
Cooper Medical College
DATE Feby 1900
NO. 38524 CLASS 0
~~GIFT OF~~
Purchase

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305

A SYSTEM OF MEDICINE

The M Co.

A

SYSTEM OF MEDICINE

BY MANY WRITERS

EDITED BY

THOMAS CLIFFORD ALLBUTT

M.A., M.D., LL.D., F.R.C.P., F.R.S., F.L.S., F.S.A.

REGIUS PROFESSOR OF PHYSIC IN THE UNIVERSITY OF CAMBRIDGE,
FELLOW OF GONVILLE AND CAIUS COLLEGE

VOLUME IV

LANE MEDICAL LIBRARY
3854
SAN FRANCISCO

New York
THE MACMILLAN COMPANY
LONDON: MACMILLAN & CO., LTD.
1898

B
All rights reserved

Set up and electrotyped August, 1897. Reprinted September, 1898.

Norwood Press:
Berwick & Smith, Norwood, Mass., U.S.A

A421
v.4
1898

PREFACE

SOME of our readers have complained of the unsystematic use of names and titles in our articles. The complaint is well founded; my reply is that I have made convenience my sole guide in this respect, and this reply I conceive to be satisfactory.

As I said in my Introduction, medical nomenclature is so backward, and for some time to come must be so backward, that it would be worse than useless to present our names under any pretence of system.

In the preparation of this volume I have had the advantage, as hitherto, of Dr. Rolleston's valuable assistance. I have also to thank Dr. Saundby for kindly revising the article on "Diabetes Insipidus," by my late friend Dr. Ralfe, who passed away in the prime of life soon after he had placed his manuscript in my hands.

The remainder of the work is in an advanced state, and will be published with as little delay as possible.

THE EDITOR.

CAMBRIDGE, *June* 1897.

CONTENTS

GENERAL DISEASES OF OBSCURE CAUSATION

PAGE

Acute Rheumatism, or Rheumatic Fever. Dr. Church and Dr. Cheadle . 1
Chronic Rheumatism. Dr. Archibald Garrod 56
Muscular Rheumatism. Dr. Archibald Garrod 61
Gonorrhœal Rheumatism. Dr. Archibald Garrod 64
Rheumatoid Arthritis. Dr. Kent Spender and Dr. Archibald Garrod . 73
Rheumatoid Arthritis in Children. Dr. Still 102
Rickets. Dr. Cheadle 108
Osteomalacia, or Mollities Ossium. Mr. Bowlby 147
Osteitis Deformans. Mr. Bowlby 149
Acromegaly. Mr. Bowlby 151
Hypertrophic Pulmonary Osteo-Arthropathy. Mr. Bowlby . . 153
Gout. Sir William Roberts 155
Diabetes Mellitus. Dr. Saundby 195
Diabetes Insipidus. Dr. Ralfe (the late) 234
Lardaceous Disease. Dr. Howship Dickinson 255

DISEASES OF ALIMENTATION AND EXCRETION

General Pathology of Digestion. Drs. Ralfe and Soltau Fenwick . . 281
General Pathology of Secretion. Dr. Rose Bradford . . . 304
Shock and Collapse. Dr. Cobbett 320
Diseases of the Mouth. Dr. W. A. Wills 333
Diseases of the Œsophagus. Dr. Rolleston 360

DISEASES OF THE STOMACH

Dyspepsia. Dr. Lauder Brunton 385
Dyspepsia in Childhood. Dr. Soltau Fenwick 407

PAGE

GASTRITIS, ETC. Dr. Lauder Brunton and Dr. Leith 415
SEA-SICKNESS. Dr. Stocker 445
MOUNTAIN-SICKNESS. Dr. Clifford Allbutt 456
NEUROSES OF THE STOMACH. Dr. Clifford Allbutt 462
DILATATION OF THE STOMACH. Dr. Clifford Allbutt 485
ULCER OF THE STOMACH AND DUODENUM. Dr. Dreschfeld . . . 517
TUMOURS OF THE STOMACH. Dr. Hale White 554

SUBPHRENIC ABSCESS. Dr. Lee Dickinson 570
DIAPHRAGMATIC HERNIA. Dr. Lee Dickinson 577
ABDOMINAL DIAGNOSIS FROM A GYNÆCOLOGICAL STANDPOINT.
Dr. Playfair 579
ENTEROPTOSIS. Mr. Treves 587

DISEASES OF THE PERITONEUM

ACUTE PERITONITIS. Mr. Treves 601
ACUTE PERITONITIS OF UNDETERMINED NATURE. Dr. Allchin . . 631
CHRONIC PERITONITIS. Dr. Allchin 638
TUBERCLE OF THE PERITONEUM. Dr. Allchin 654
NEW GROWTHS OF THE PERITONEUM. Dr. Allchin 672

DISEASES OF THE BOWELS

PHYSIOLOGY OF FÆCAL EVACUATION. Dr. Lauder Brunton . . . 693
DISEASES OF THE SMALL INTESTINE. H. D. Rolleston . . . 709
COLIC. Dr. Hale White 727
DIARRHŒA. Dr. Lauder Brunton 733
DIARRHŒAS OF CHILDREN. Dr. Eustace Smith 746
SPRUE, OR PSILOSIS. Dr. Patrick Manson 774
INTESTINAL OBSTRUCTION. Mr. Treves 794
PERITYPHLITIS. Mr. Treves 879
DISEASES OF THE COLON. Dr. Hale White 937
THE DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE ANUS AND RECTUM.
Dr. Allingham 979

INDEXES 985

ILLUSTRATIONS

FIG.		PAGE
1.	Acute Rheumatism, with numerous large Nodules	46
2.	Section of Subcutaneous Tendinous Nodule in Acute Rheumatism	48
3.	Section of Aortic Valve in Ulcerative Endocarditis in a Child	49
4.	Chronic Arthritis with enlargement of Lymphatic Glands and Spleen	103
5.	Rickets, showing general Deformity and Distortion of the Bony Skeleton, Muscular Wasting, and Debility	109
6.	Rickets, showing contraction of Thorax and prominence of Abdomen	110
7.	Rickets, a case of extreme bossing of the Skull	113
8.	Rickets, Lateral View of the same Skull	114
9.	Rickets, showing beading of Ribs, transverse Groove, and lateral depression of Thorax	115
10.	Vertical Section through upper half of Humerus of a child aged one year	117
11.	Part of other half of same Bone as that shown in Fig. 10	119
12.	Late Rickets	125
13.	Abdominal Swelling caused by Lardaceous Liver	261
14.	Liver as made out by touch	261
15.	Enlargement of Liver from Lardaceous Disease of Syphilitic Origin	262
16.	The Mucous and part of Submucous Coats in Phlegmonous Gastritis	424
17.	The Submucous and other Coats ,, ,,	425
18.	The Muscular Coat ,, ,,	426
19.	Normal Position of the full Stomach	486
20.	Diagram of Stomach in which Pyloric Obstruction was produced by Kink caused by Adhesion to Gall-bladder	487
21.	Enteroptosis: Displacement of Stomach, Liver, and Spleen	490
22.	Outline of Stomach: Atonic and Obstructive Dilatation	496
23.	Obstructive Dilatation—Non-malignant Stenosis of Pylorus	503

PLATE

PAGE

Section of Liver and Small Intestine in a case of Extreme Rickets and Tetany . 145

CHARTS

No.

1. Cases of Rheumatic Fever in London Hospital, 1873-81 . . . 8
2. ,, ,, St. Bartholomew's Hospital, 1882-93 . . 8
3. Incidence of Enteric Fever in London for twelve months . . . 9

Charts showing Temperature in Acute Peritonitis . . *To face page* 620

LIST OF AUTHORS

Allbutt, Thomas Clifford, M.D., LL.D., F.R.C.P., F.R.S., Regius Professor of Physic in the University of Cambridge, Fellow of Gonville and Caius College, Consulting Physician to the Leeds General Infirmary.

Allchin, W. H., M.D., F.R.C.P., Senior Physician to the Westminster Hospital, and Lecturer on Clinical Medicine.

Allingham, Herbert Wm., F.R.C.S., Surgeon to the Great Northern Hospital; Assistant Surgeon to St. George's Hospital.

Bowlby, Anthony A., F.R.C.S., Assistant Surgeon and Surgeon to Throat Department, St. Bartholomew's Hospital.

Bradford, John Rose, M.D., D.Sc., F.R.C.P., F.R.S., Physician to University College Hospital; Professor of Materia Medica and Therapeutics, University College, London; Professor Superintendent of the Brown Institution, London.

Brunton, T. Lauder, M.D., LL.D., D.Sc., F.R.C.P., F.R.S., Physician to St. Bartholomew's Hospital, and Lecturer on Pharmacology and Therapeutics.

Cheadle, W. B., M.D., F.R.C.P., Physician and Lecturer on Clinical Medicine, St. Mary's Hospital; Consulting Physician, Hospital for Sick Children.

Church, W. S., M.D., F.R.C.P., Physician to St. Bartholomew's Hospital.

Cobbett, Louis, M.D., F.R.C.S., John Lucas Walker Student of Pathology in the University of Cambridge.

Dickinson, W. Lee, M.D., F.R.C.P., Assistant Physician to St. George's Hospital and to the Hospital for Sick Children.

Dickinson, W. Howship, M.D., F.R.C.P., Consulting Physician, St. George's Hospital and Hospital for Sick Children; Honorary Fellow of Gonville and Caius College, Cambridge.

Dreschfeld, Julius, M.D., B.Sc., F.R.C.P., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Victoria University.

Fenwick, W. Soltau, M.D., M.R.C.P., Physician to Out-patients Evelina Hospital for Sick Children; Physician, London Temperance Hospital.

Garrod, Archibald E., M.D., F.R.C.P., Medical Registrar and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; Assistant Physician to the Hospital for Sick Children.

Leith, R. F. C., M.B., C.M., B.Sc., M.A., F.R.C.P.E., Lecturer on Pathology and Morbid Anatomy, Edinburgh Medical School; and Pathologist to the Royal Infirmary, Edinburgh.

Manson, Patrick, M.D., LL.D., F.R.C.P., Physician to the Seamen's Hospital, Albert Dock Branch; Lecturer on Tropical Diseases to St. George's and Charing Cross Hospitals.

Playfair, W. S., M.D., LL.D., F.R.C.P., Professor of Obstetric Medicine in King's College, and Obstetric Physician to King's College Hospital.

Ralfe, Charles Henry (the late), M.D., F.R.C.P., sometime Physician to the London Hospital and to the Seamen's Hospital, Greenwich.

Roberts, Sir William, M.D., F.R.C.P., F.R.S., formerly Physician to the Royal Infirmary, Manchester, and Professor of Medicine, Victoria University.

Rolleston, Humphry Davy, M.D., F.R.C.P., late Fellow of St. John's College, Cambridge; Assistant Physician and Lecturer on Pathology to St. George's Hospital; Assistant Physician to the Victoria Hospital for Children.

Saundby, R., M.D., F.R.C.P., Physician to the General Hospital, and Professor of Medicine, Mason College, Birmingham.

Smith, Eustace, M.D., F.R.C.P., Physician to the East London Hospital for Children, and to the City of London Hospital for Diseases of the Chest.

Spender, J. Kent, M.D., late Physician Royal Mineral Water Hospital, Bath.

Still, George F., M.D., M.R.C.P., Medical Registrar and Pathologist, the Hospital for Sick Children, Great Ormond Street.

Stocker, J. R., M.B., M.R.C.P., Medical Officer, Board of Trade, Glasgow.

Treves, Frederick, F.R.C.S., Surgeon and Lecturer on Surgery to the London Hospital; Surgeon in Ordinary to H.R.H. the Duke of York.

White, W., Hale, M.D., F.R.C.P., Physician and Lecturer on Pharmacology and Therapeutics to Guy's Hospital.

Wills, W. A., M.D., M.R.C.P., Assistant Physician and Lecturer on Elementary Medicine to the Westminster Hospital; Physician, North-Eastern Hospital for Children.

ERRATUM

VOL. II.

Page xiii., line 2, *for* "Westminster" *read* "West London."

In order to avoid frequent interruption of the text, the Editor has only inserted the numbers indicative of items in the lists of "References" in cases of emphasis, where two or more references to one author are in the list, where an author is quoted from a work published under another name, or where an authoritative statement is made without mention of the author's name. In ordinary cases an author's name is a sufficient indication of the corresponding item in the list.

GENERAL DISEASES OF OBSCURE CAUSATION

ACUTE RHEUMATISM, OR RHEUMATIC FEVER

ACUTE Rheumatism, or Rheumatic Fever, is a disease more easily described than defined. No line of separation can be drawn between the cases classified as subacute and acute; and in this article acute rheumatism will be considered as equivalent to a synovitis accompanied by pyrexia, and generally multiple.

Under the name rheumatism is included a vast medley of pathological conditions having little or no connection with each other, except in the presence of pain in the muscles or about the joints. The word, at first explanatory of the pathology of the morbid condition (the pain being attributed to rheum[1] flowing down from the brain and settling in the affected part), has now become a convenient term for embracing myalgic, neurotic, and arthritic pain dependent on very various causes of whose nature we are often ignorant.[2] The inclusion of three very distinct affections—gout, osteoarthritis, and gonorrhœal rheumatism—under the head of acute rheumatism vitiates to a great extent all the statistics of the disease given by the older writers.

Sydenham separated acute rheumatism from gout (71), giving an excellent clinical description of the disease, and remarking that "it is commonest in autumn, chiefly attacking the young and vigorous"; he goes on to say that it very rarely kills the patient. The mortality during an acute attack is very small, and of those who die in the course of the attack, death, excepting in cases of hyperpyrexia, is almost invariably due to secondary lesions, such as peri- or endocarditis or, more rarely, pneumonia or pleurisy. I say almost invariably, for in very rare instances death occurs suddenly without hyperpyrexia or any discoverable injury to the lungs or heart. One such case I have seen myself. A girl aged nineteen, a nursemaid, was taken ill on May 25th, 1870, with pain in the right foot, followed by pain in the legs and hands; the joints were swollen and very painful. She was admitted to St. Bartholomew's Hospital on June 1st. She was fairly well nourished; besides pain in her limbs

[1] Jejuna, vigila, caleas dape, tuque labora,
Inspira calidum, modicum bibe, comprime flatum,
Haec bene tu serva si vis depellere rheuma.
Si fluat ad pectus dicatur rheuma catarrhus;
Si ad fauces, bronchus; si ad nares, esto coryza.
Schola Salernitana, cap. lxxxii.

[2] Ballonius is credited with being the first to use the word rheumatismal in its present sense. *De rheumatismo et pleuritide*, 1642.

she complained of some discomfort at the epigastrium; her tongue was coated, appetite gone, bowels costive; there was some vomiting; the urine was high-coloured, alkaline, free from albumin; skin moist; left knee much swollen. States that she has had very little sleep; pulse 108, good volume, heart sounds natural, respiration normal, temperature 99·5°. During the afternoon of the day of admission she was very restless, and complained of increasing pain at the epigastrium and in the præcordial region. At 9.30 P.M. she suddenly became very pale, sick and pulseless, with pupils widely dilated, and died at 10.15 P.M.[1] Nothing abnormal was found in any organ except very slight beading along the margin of the mitral valve. Senator[2] speaks of sudden death occurring in "paroxysms of palpitation and oppression" not dependent on cardiac complications.

Prevalence.—Taking the statistics of the British Army, which consists for the most part of men at the period of life most subject to acute rheumatism, I find that the deaths ascribed to rheumatic fever in the years 1889-91 amounted to twenty-seven; that is to say, 1·01 in every 25,000. In the Native Army of India the death-rate is still lower, being 0·51 in every 25,000. Among our general population the Registrar-General's returns for the five years 1881-85 give ninety-seven per million living as the death-rate from this disease; in the five succeeding years (1886-91) the rate had fallen to eighty-nine per million living. The mortality from the disease, as given in the Registrar-General's returns, is therefore no measure of its frequency, nor of the injury inflicted by it; a very large proportion of the deaths attributed to heart disease being due to lesions caused by acute rheumatism. Rheumatic fever is a ubiquitous disease, and is met with from arctic to torrid zones; but it is more frequent in subtropical and temperate climes. No race of men is exempt, although Europeans in the subtropical and warmer temperate regions appear to suffer more frequently than the natives.

Mortality.—The actual mortality caused directly by rheumatic fever varies but little in the returns I have been able to consult. I find that in our own Registrar-General's returns the average number of deaths ascribed to rheumatic fever and rheumatism of the heart for the six years 1887-92 is 4·67 per thousand of deaths. In Scotland from the returns of mortality in the eight principal towns for the last seven years I find it 4·97. In Ireland it is 2·30,[3] in Paris 4·29,[4] in New South Wales 3·23,[5] in Queensland 3·02,[6] in New York City and Brooklyn 3·48[7] per thousand of deaths from all causes.

[1] See also a somewhat similar case of Dr. Goodhart's (41).

[2] Senator (67) refers also to Rathery's case 62, but on turning to his reference I find it was a case of a girl aged three, who was seized with dyspnœa and became cyanosed, and died ten hours later. Post-mortem, pleurisy and collapse of lung were found, and ante-mortem clot in the right ventricle.

[3] Average for 8 years.

[4] ,, ,, 3 for rheumatism, rheumatic fever not being separated.

[5] ,, ,, 5 ,,

[6] ,, ,, 5 ,,

[7] ,, ,, 6 ,,

Etiology and geographical distribution of rheumatic fever.—In our present ignorance of the immediate causes of the disease, it appears to be useless to discuss at any length the various hypotheses which have ascribed it to the presence in the blood or tissues of an undue amount of some of the products of chemical change in our bodies, or to the formation of chemical compounds, not usually present, under the influence of fatigue, chill, or some other unfavourable condition. The advocates of what has been termed the nervous origin of rheumatic fever have not explained in what way the central nervous system becomes affected; for it is indubitable that many cases occur without any discoverable irritation of the peripheral nerves. Although no satisfactory evidence has as yet been procured of the constant presence of any single specific micro-organism, yet in not a few of its features the whole course of rheumatic fever resembles an infective disease; moreover the curves of its mortality statistics in this country approximate very closely to those of pyæmia, puerperal fever, and erysipelas, diseases which are certainly associated with specific micro-organisms. The undoubted connection of ulcerative endocarditis—which until recent years was not recognised as distinct from simple rheumatic disease of the valves of the heart—with pyæmia and puerperal fever throws some doubt on the correctness of the curves given by Longstaff from statistics which began nearly forty years ago.

The similarity which acute rheumatism bears in many of its features to gout led to *the chemical theory* of its production; a theory supported by many observers in this country. Prout is said by Fuller to have been the first to throw out the suggestion that it is due to lactic acid; and this view, further amplified by Todd, was accepted by Fuller as the most probable of all the theories up to that time propounded. The experiments of Richardson were discredited by the more extended ones of Rayher; and the observations of Foster and of Kulz on the occurrenee of articular pain and swelling in diabetics under treatment by lactic acid have not been confirmed by later experience. Dr. Latham in his Croonian Lectures considered the exciting cause of rheumatism to be the presence of lactic acid together with uric acid, the latter acting as an irritant to the nervous centres and to the subsidiary ganglia in connection with them; and Haig (44) suggests that any cause producing pyrexia may determine the precipitation of uric acid in the joints, and thus give rise to the arthritic symptoms. It is difficult to believe that uric acid can give rise in our bodies to such very different pathological conditions as those met with in gout and acute rheumatism: the similarity is superficial whilst the whole pathological and clinical course is different. If the evidence in favour of the chemical theory of acute rheumatism be unsatisfactory, the same may be said of the nervous theory first suggested by Dr. J. K. Mitchell sixty years ago, and advocated by Canstatt, Day, and others; for neither the nutritional lesions which occur in and about the articulations in connection with spinal degenerative changes, nor those which follow injury

to or division of the nerves supplying the joints, resemble the conditions met with in acute rheumatism (8).

The curves formed by the statistics not only of the mortality of acute rheumatism, but also of its occurrence, are very similar to those of the infective diseases. I have already stated that the mortality curve given by Longstaff is not to be relied upon, as deaths from ulcerative endocarditis are included in it. Babes has drawn attention to what he calls "bacterial associations"; and has made the rather alarming observation that "among 350 hospital patients with non-infectious diseases I could hardly find fifty who did not suffer from bacterial associations, and a great part of the patients with non-infectious diseases died in consequence of these bacterial associations." The fact that a considerable number of the deaths from ulcerative endocarditis occur in persons who have suffered from valvular disease of rheumatic origin leads one to think that there may be more truth in Babes' statement than one is willing to believe.

Many observers have found *micro-organisms* in acute rheumatism, but in most instances they have been staphylococci, streptococci, or other organisms belonging to the septic or putrefactive class (56).[1] If the statement of Babes be correct, that "there are months in which all deaths in hospital show association with streptococci or with staphylococcus aureus," these organisms would naturally be found in the non-fatal cases as well.

Garrod (37) has constructed charts, from the statistics of Lange in Copenhagen and Gabbett in London, showing the curves during a series of years yielded by first attacks of rheumatic fever; *atmospheric and climatic causes* may influence these curves, but no satisfactory evidence has yet proved any connection between rainfall, low temperature, or sudden changes in this or other atmospheric conditions, and the prevalence of the disease. Hirsch, from the absence of any constant meteorological influences, and the remarkable fluctuations in the prevalence of the disease at the several points which have been carefully watched, considers that "rheumatic fever now seems to have an assured place among the acute infective diseases." In the Milroy Lectures of 1895, Dr. Newsholme has brought forward a large amount of statistical evidence to support the theory that acute rheumatism is one of the infectious diseases. He showed that the rates both of the mortality and of the frequency of the disease fluctuate in a manner very similar to those of such infectious diseases as scarlatina and erysipelas.

Very interesting observations have been made on the frequency with which rheumatic fever occurs in *particular houses*, which tend to support the infectious theory of the disease. Dr. Friedlander[2] of Leipzig saw twelve cases from the same house in three years; and during another three years met with eighteen cases from two houses. Edlefsen, at

[1] Dr. Mantle describes a bacillus as present in the effusion in the joints and in the blood. His observations have not been confirmed.

[2] Quoted by Dr. Newsholme in the Milroy Lectures, 1895.

Kiel, reports that of 728 cases of rheumatic fever occurring in 492 houses, he met with

2	cases in	one house	100	times.
3	"	"	27	"
4	"	"	5	"
5	"	"	5	"
6	"	"	1	"
7	"	"	1	"

Drs. Fiessinger, Mantle, and Dalton have also recorded the occurrence of groups of cases arising within short periods in the same house.

The British Army, consisting of young men at a time of life prone to the disease, and placed under similar conditions as to hygiene, food, and work under very various meteorological conditions, affords excellent material for comparison. I have abstracted (*vide* Appendix I.) the returns of rheumatic fever from the different stations during the last ten years to see if climatic conditions appeared to have any connection with the prevalence of the disease. I have also collected (*vide* Appendix II.) the official returns from our colonial dependencies showing the ratios which admissions to hospital for rheumatic fever bear to admissions from all causes. An examination of both the military and colonial returns bears out the accepted view that rheumatic fever is comparatively rare in tropical countries; whilst its distribution in the subtropical and temperate zones is very capricious, and in no way dependent on the mean temperature of the locality. Its prevalence at Mauritius, which is just within the tropics, appears as an exception; but it must be remembered that the numbers of our troops there are small, and that the proportion of the rheumatic fever cases in the civil hospitals is not equally high.

That *racial differences* have some bearing on the question is shown by the fact that whilst the average number of attacks per thousand among our British troops in India is larger than amongst the same troops when quartered in Scotland and Ireland, the ratio given by our Native Indian Army is the lowest of all.

That *chill* is probably the most important factor in determining an attack of rheumatic fever is not only borne out by general experience, but to a certain extent by the geographical distribution of the disease. Contrary to what is usually believed, high and dry lands, where the temperature varies between wide limits in the twenty-four hours, appear to be particularly conducive to rheumatic fever. Thus we find it of comparatively frequent occurrence in Egypt, the Cape, Central Arabia, and the high elevated plateaus of Bavaria and Mexico (46); whilst in lower and moister localities, of a more even but equally high temperature, it is infrequent. The great fall of temperature which occurs during some months of the year after sunset at our Mediterranean stations may account for the high averages yielded by them in our military statistics; but it must also be borne in mind that Malta[1] fever [*vide* vol. ii. p.

[1] Dr. Dalton refers to the frequency of the disease at Malta, as possibly strengthening his view that it may be caused by fæcal and sewer emanations (18).

463] is accompanied by severe arthritic pains; hence cases not truly rheumatic may be included in the statistics and raise the average. Unfortunately the military returns during the Crimean War do not afford data for comparing the proportion of cases of rheumatic fever with those of rheumatism. Our troops in the Crimean War were exposed to damp and great cold as well as fatigue. The only information I find bearing on this point is the statement by Dr. Lyons: "Rheumatic pericarditis seems to have been very rare during our army's occupation in the Crimea, notwithstanding the rigour of the climate."

The influence of fatigue and exposure in a dry and otherwise salubrious climate is shown very prominently by the military returns during the Egyptian and Cape Wars. In Egypt, during the years that our troops were on active service in the field, the average number of admissions for rheumatic fever reached the high ratio of 29·68 per 1000 men; and in the Cape during the years of the Kaffir War an increase of over 5 per cent in the cases of rheumatic fever was seen.

The United Kingdom, and England in particular, is regarded by many writers, especially on the Continent, as pre-eminently the country of acute rheumatism. It is almost impossible to form any opinion as to the frequency of its occurrence in the United Kingdom. I have arranged in the Appendices V. and VI. all the returns that I have obtained from the metropolitan and provincial hospitals. The returns are very imperfect, and of less value than I had hoped; whether as indicating the proportion rheumatic fever bears to other diseases, or as showing the effect of position, climate, nature of the soil, or character of the population on the occurrence of the disease. How little reliance can be placed on these returns as giving information regarding the frequency of the disease in different localities may be seen from the returns of hospitals in the same town, which differ widely in the proportionate number of cases to the sum total of the medical cases treated: for instance, there is a difference of 2·40 per cent between the returns of the Royal Infirmary and the Royal Southern Hospital, Liverpool; and a still greater difference between the returns of the Salford Royal Hospital and the Manchester Royal Infirmary.

The population of our large manufacturing centres appears to suffer more than that of our rural districts; but this may be due to the larger proportion of cases in country districts treated in their own homes. The returns from the East Coast are lower than those from the West; but with the exception of Newcastle there are no returns from large towns, such as Hull and Hartlepool. The prevalence of the disease in the Isle of Man and Whitehaven is remarkable.

Scotland suffers less than England, corroborating the evidence given by the military returns; and there is no proof that the disease is less frequent, as has been stated, in Cornwall,[1] Guernsey, and the Isle of Wight than in many other parts of the United Kingdom of equal size. Senator

[1] I find that in the ten years 1882-91, 235 deaths in Cornwall are ascribed to rheumatic fever and rheumatism of the heart; and a correspondent who practises at Redruth says: "Cases of acute rheumatism are not infrequent in my own practice."

quotes London as having the highest percentage of any European city; and gives its average as 11·5 per cent of the sum total of disease. This average very greatly exceeds that now found in the statistics of the London hospitals; and I find that an examination of the cases treated in the clinics of the physicians of the Charité Hospital, Berlin, during the years 1889-94 give very much the same percentage of cases of rheumatic fever (4·87) as those yielded by the medical statistics of St. Bartholomew's Hospital. Rheumatic fever, as Hirsch has remarked, is "ubiquitous, and is more evenly spread over the world than is ordinarily stated."

Influence of seasons (37).—That seasonal changes play some part in the prevalence of rheumatic fever is undoubted. All the collected statistics show fluctuations in the curves of its frequency during the various months of the year, which appear fairly constant in the localities where the statistics are compiled, but present great discrepancies when compared with those drawn up in other places. Dr. Newsholme attributes the prevalence of rheumatic fever at different times to the influence of ground water. He considers that low ground water is an indication of certain conditions of dryness and temperature of the subsoil favourable to the growth of the telluric contagium of rheumatic fever; whilst admitting that low ground water unassociated with the other necessary conditions may not be productive of rheumatic fever, he contends that we never find excess or prevalence of rheumatic fever with a high level of ground water. Somewhat similar conclusions have been arrived at by Dr. L. Weber. In London the old and widespread belief that the disease is most common in the autumn appears well founded.

Dr. Gabbett tabulated 2000 cases of rheumatic fever, admitted to the London Hospital during the nine years 1873-81, according to the months; and I have arranged his figures in the form of a chart for comparison with that drawn up by Mr. L. C. P. Phillips of 1998 cases at St. Bartholomew's Hospital during the twelve years 1882-93.

It will be seen that in each series the curve made by the numbers of first attacks is identical with that made by the whole number of patients. It will also be noted that the curves made by Dr. Gabbett's figures are almost identical with those at St. Bartholomew's Hospital during a period subsequent to the nine years tabulated by Dr. Gabbett; the only difference is that at the London Hospital the maximum is reached in November, whereas in Mr. Phillip's chart the maximum is found in October: this difference is probably due to the London Hospital chart dealing with the admissions, whilst that of St. Bartholomew's deals with the onset of the symptoms.

The monthly curve of the incidence of rheumatic fever on the metropolitan population is remarkably similar to that of enteric fever. In both diseases the number of attacks falls during January, and is at its lowest during February, March, April, May, and June; then enteric fever begins to rise, and goes up with a bound during July and August, attaining its maximum in September and October, and then rapidly sinking. Rheumatic fever does not rise in numbers until later

n the year, when it leaps up and attains its maximum in October, and begins to subside again. The constancy of these seasonal variations

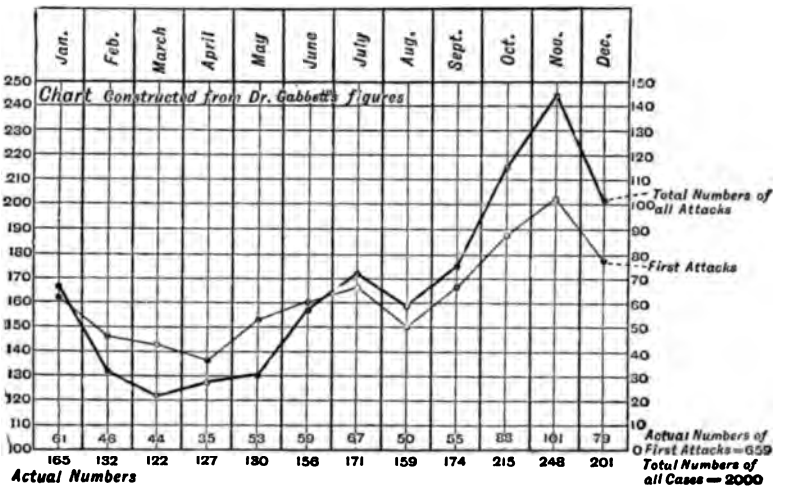


CHART 1.—Cases of rheumatic fever in London Hospital 1873-81.

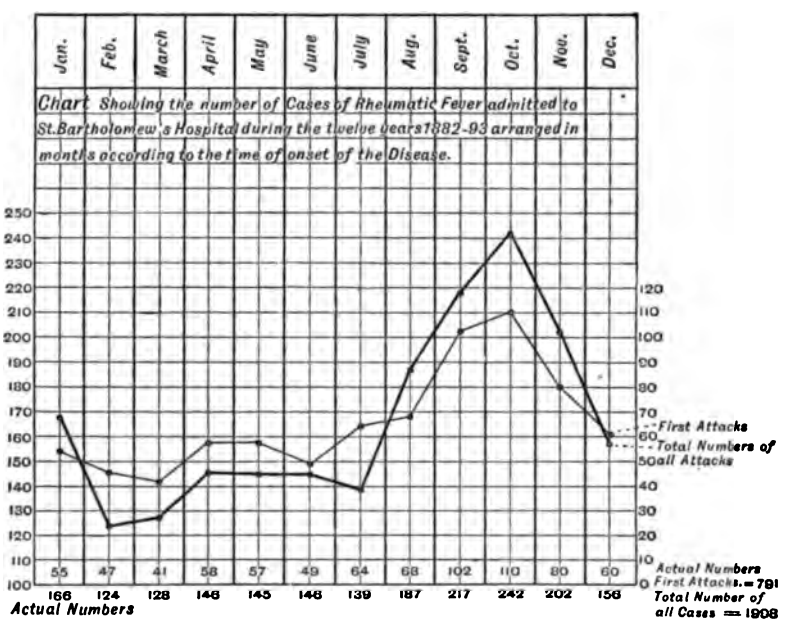


CHART 2.—Cases of rheumatic fever in St. Bartholomew's Hospital 1882-93.

lends probability to the infective theory of rheumatic fever, and supports the view that its immediate cause is a micro-organism capable of multipl-

cation in a suitable nidus outside our bodies. The constancy of the seasonal curve seems to be opposed to the theory advanced by Dr. Newsholme of a connection between the prevalence of rheumatic fever and a low level of ground water. I cannot think that London, where the bulk of the rain that falls runs off on the surface, and where the subsoil is drained by railway tunnels and cuttings and sewers, can have any great variations in the level of the ground water; or that for twenty-one years it should vary so uniformly.

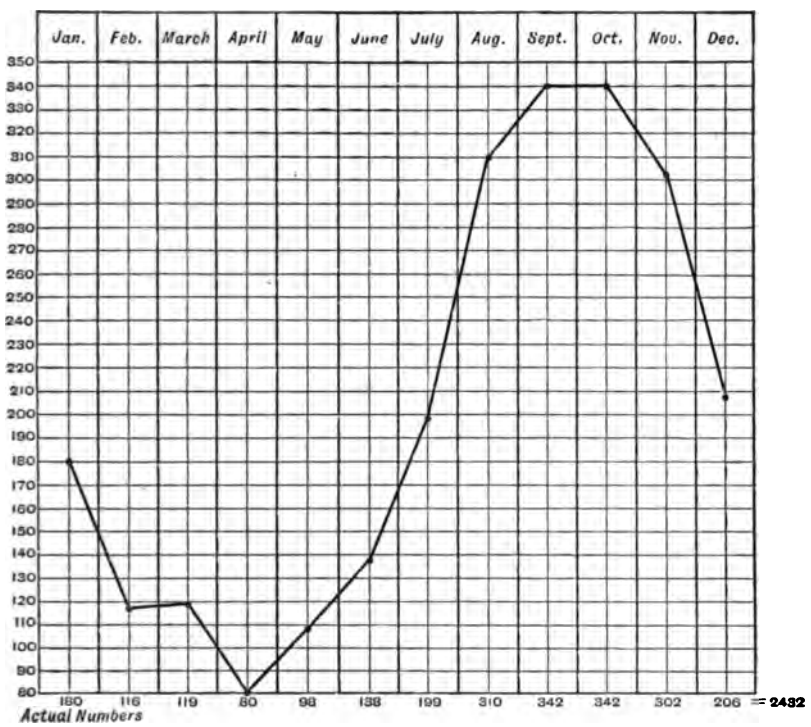


CHART 3.—Incidence of enteric fever in London for twelve months.

Sex (37).—It is generally stated that acute rheumatism is more prevalent in men than in women, owing to their greater liability to exposure in the course of their work. This explanation of the greater frequency of the disease in men, if it exists, cannot be maintained. In London, if not elsewhere, the disease is as common, if not commoner, among indoor workers as among those following outdoor employments; and the figures given above show that it is most prevalent at a season of the year when the weather is not usually severe, and is least prevalent during February, March, and April, the three months in which outdoor workers here are exposed to great vicissitudes of temperature and weather. The relative frequency of the disease in the sexes is an extremely difficult fact to

ascertain; the statistics of various writers differ very widely. At St. Bartholomew's, and at most of the other metropolitan hospitals whose figures I have gone through, the numbers of the two sexes are nearly the same, the excess being slightly on the side of the men; and it must be remembered that in most hospitals the medical beds for men exceed in number those for women. Women are particularly liable to the disease when exhausted by parturition and prolonged lactation; but the numbers given in the older statistics must be received with caution, as many cases of puerperal septicæmia or pyæmia are included, and a considerable fraction of the alleged acute rheumatic attacks are not true rheumatic fever, but the acute stage of chronic osteoarthritis.

Age.—In considering the influence of age on the disease first attacks only should be regarded; for the tendency of rheumatic patients to frequent attacks at short intervals falsifies any deductions which can be made from the general statistics of the disease. Acute rheumatism is essentially a disease of adolescence and early man and womanhood. Of 253 instances of first attacks of which I have records—

16	were under 10 years of age	= 6·32	per cent.
100	between 10 and 20 years of age	= 39·52	,,
85	,, 20 ,, 30 ,,	= 33·59	,,
47	,, 30 ,, 40 ,,	= 18·57	,,
2	,, 40 ,, 50 ,,	= 0·79	,,
2	,, 50 ,, 60 ,,	= 0·79	,,
1	age not known.		
253			

These figures show the greater liability of youth and early adult life. Still no period of life is exempt; the disease has been described in a baby of twenty-three days, and in another of four weeks (67); and a first attack from which a perfect recovery was made is reported in a man over eighty (15).

Heredity.—That some families are predisposed to the disease is shown by the frequency with which we see several children in the same family affected sooner or later by it. Dr. Cheadle is of opinion that the tendency to rheumatism is transmitted as strongly as the tendency to gout; and that hereditary predisposition is found in 20 per cent of rheumatic children. Dr. L. Weber (77) uses the recurrence of rheumatic fever in several members of the same family as an argument in favour of its being "a house disease, the underground of houses in certain localities being infiltrated with the virus, which will be set free after prolonged dryness." The occurrence of rheumatic fever in phthisical subjects is not very uncommon. I have met with families in whom there appeared to be a very strong proclivity to tubercular disease and rheumatic fever; and with one in which nearly all the children suffered either from rheumatic fever or tubercular disease of the lungs. I have also known several instances of tubercular phthisis, beginning shortly after an attack of

rheumatic fever[1] associated with severe valvular mischief, in which I had no doubt that the tubercular disease was really subsequent to the attack of rheumatism. Statistics of heredity, where we have to deal with an ill-defined disease, easily confounded with gout and rheumatism, appear to me to have little value, and those drawn up from hospital patients to be quite worthless;[2] that the disease is hereditary in the same way as gout or insanity seems to me to require further evidence.

Symptoms.—*The onset* of rheumatic fever is in most cases gradual; a few days of malaise accompanied by pains in the limbs and frequently associated with slight sore throat is a common mode: well-marked rigors are unusual, but chilliness is often complained of; the temperature is raised; appetite is lost; the tongue becomes furred, often thickly coated, in severe cases the tip and edges may be red, but in milder cases are pallid and the whole organ flabby. Pain usually begins in one of the larger joints—the knee, hip, elbow, ankle or shoulder.

With the appearance of pain and swelling in the joints the fever increases; the temperature may rise to 103° F. or more, but in hospital practice the vast majority of patients when admitted have a temperature varying between 101° and 103° F. The face is flushed; the pulse becomes more frequent; thirst increases, and there is complete anorexia; constipation is usually present. The severity of the pain prevents sleep; delirium even in severe cases is rare. The joint or joints first affected may remain the seat of pain or recover; but by the third or fourth day from the beginning of the illness pain and swelling have commonly attacked several joints in succession, the pain passing off in one as it increases in another: in this way nearly all the larger joints of the extremities may be affected; more rarely the sterno-clavicular and intervertebral joints also suffer.

One of the most striking features of the disease is the condition of the skin. Notwithstanding the pyrexia, the skin does not feel hot to the touch; excepting in hyperpyrexia, it is moist and perspiring, and in by far the greater number of cases the sweating is excessive. The urine from the first is high-coloured, scanty, of high specific gravity, strongly acid, and deposits on cooling a copious precipitate of urates; not infrequently it contains crystals of uric acid. When the fever is at its height a small quantity of albumin is often present in it. The *duration* of the fever is variable; in previously healthy young adults, provided they escape pericarditis or severe endocarditis, the acute symptoms, when uninfluenced by drugs, frequently subside in eight or nine days, and convalescence is established in another ten days.[3]

Such, very briefly, is the clinical aspect of the onset and course of

[1] Dr. J. A. Austen (3) published four cases of rheumatic fever followed by lung disease, but it is not clear that in his cases the tubercular disease of the lung was not in existence before the rheumatism.

[2] For the relative frequency of rheumatic fever in the families of rheumatic and other patients, *vide* (37).

[3] *Vide* Sir William Gull and Dr. Sutton's paper (43).

rheumatic fever of moderate severity, unaccompanied by either cardiac or pulmonary complications. It is necessary, however, to examine a little more closely into its course and the symptoms presented.

Though the onset is usually gradual, it is occasionally quite sudden. I have known it appear with excruciating pain coming on suddenly in a joint, and incapacitating the sufferer from walking home. When rigors occur they are often frequent and of moderate severity. Whilst the duration of the fever in uncomplicated and favourable cases may be as above stated, much more commonly it runs a protracted course, the acute symptoms subsiding and recrudescing again and again. The occurrence of genuine *relapses* is also common under any form of treatment; among my own 859 cases 127, or 14·78 per cent, had relapses[1] which occurred with nearly equal frequency in first and second attacks, but were less often met with in third or subsequent ones. Age, under forty, appears to have little influence; as in

34	patients under	10	years of age	5	relapsed	= 17·64	per cent.
315	„	20	„	46	„	= 14·60	„
297	„	30	„	43	„	= 14·47	„
140	„	40	„	29	„	= 20·71	„
46	„	50	„	3	„	= 6·52	„
27	„ over 50 or age not stated,			1	„	= 3·70	„

The average stay of patients in hospital is a very imperfect guide to the duration of the disease; a few prolonged cases greatly increase the average of the whole. I find that for my own cases the average stay has been thirty-two days, and that neither age nor the occurrence of previous attacks has much effect in increasing or reducing the length of their stay in hospital.

The *temperature* rises rapidly with the joint pains. Wunderlich truly remarks that "in an overwhelming majority of hospital cases the maximum temperature is reached either on the day of admission or almost directly after." My observation completely agrees with this, and there can be but little doubt that it is due to the fatigue and distress caused to the sufferers by their removal to hospital; 499 had in the evening of the day of admission a temperature between 101° and 103° F.; in 142 the temperature was between 103° and 104°, and in only twenty-three did it exceed 104°. In the large majority of the cases these were the highest temperatures recorded during their attack. The temperature, excepting in cases of hyperpyrexia, is highest in the evening. The advent of pericarditis or severe endocarditis is generally, though by no means invariably, marked by a rise in temperature; and the same may be said of pneumonia or pleurisy. When treated with salicylates the temperature of the patients has seldom remained above the normal

[1] This average of relapses is considerably lower than that given as occurring in Guy's Hospital in cases treated with salicylates or its allies (26). This may be due to my not considering a mere recurrence of pain without a rise in temperature as a relapse.

after the fourth or fifth day in hospital, and in a large number of the cases it sinks to normal in from forty-eight to seventy-two hours.

Sleeplessness, which is very commonly complained of, results rather from the severity of the pain in the joints than from mental excitability; and as soon as the pain is relieved complaints of want of sleep are seldom made. Delirium is exceptional; in those who have been guilty of alcoholic excesses one occasionally meets with a condition resembling delirium tremens. In a few cases, chiefly among women, the salicylate treatment pushed too freely has produced some slight wandering of mind.

Joints. — The affected joints are swollen, the skin over them has a florid blush, and serous exudation takes place into their cavities, into the tissues surrounding them, or into both. Even when neither redness nor swelling is perceptible, so long as a joint is in pain increased heat can usually be perceived by the hand laid on it. When in pain the joints are kept by the sufferers in characteristic positions: the knees slightly flexed, the ankles extended, the elbows flexed, the wrists extended, and the fingers of the hand slightly turned towards the ulnar side of the limb. One of the most characteristic features of the disease is the migratory nature of the joint affection. The joint which one day is swollen, red, and acutely painful may be found on the following day free from pain and only a little stiff on movement; whilst others, normal on the preceding day, are now the seat of swelling and violent pain. The swollen tissues round the joint seldom pit on pressure, and desquamation of the epidermis does not occur, as is so frequently the case after acute gout. Opportunities for examining joints which at the time of death were the seat of acute rheumatic inflammation are so infrequent that we can say little as to their pathological condition. In the larger joints remains of a serous effusion and occasionally flakes of lymph are found; and less frequently a layer of lymph has been observed on the synovial membrane. Dr. Moxon (26) found the sheath of the extensor tendons of the wrist full of opaque serum and masses of greenish yellow lymph; and Dr. Vivian Poore has described a very similar condition in the joints of a patient who died of hyperpyrexia. The cases of suppuration of the joints described by the older pathologists we now know were due in most instances to pyæmia, in some to gonorrhœal rheumatism.

The condition of the *skin* is remarkable, the patients sweat most profusely; even now, when it is no longer the custom to heap unnecessary blankets on the perspiring sufferers, excessive sweating is one of their most frequent complaints. Beads of perspiration collect on the forehead and temples, and run down into the eyes, adding to their discomfort; and the extremities perspire as profusely as the head and body. Very different statements have been made as to the chemical reaction of *the perspiration*. It is commonly said to have a sour smell and acid reaction; of the sour smell about rheumatic patients there can be no doubt, and that it is due to excessive perspiration is equally true; but that this is caused by any abnormal constituents in the perspiration is not proved: it is due, I believe, to fermentative changes which take place

in the perspiration after it is poured out. Exactly the same smell is present about persons in perfect health who do not change their underclothes after free perspiration induced by exercise and physical labour. Normal sweat is such a complex substance, and differs so greatly with the parts of the body where it is secreted, that little value is to be attached to observations recorded in rheumatic fever; according to my own experience, based upon a large number of observations made upon carefully cleaned skin; the perspiration on the face and chest is usually feebly acid, as it is under ordinary circumstances in health.

In connection with the excessive sweating sudamina (14) are of frequent occurrence; they are essentially the same as those which occur in scarlatina, typhoid, and other febrile states accompanied by free sweating; but they do not run quite the same course. In rheumatism they go through changes rarely if ever seen in other conditions. The vesicles, at first clear, and in fact only visible in certain lights, become much more perceptible, their contents become milky and opaque, and their bases are surrounded by a minute ring of redness. Senator, speaking of sudamina, says "they are often associated with miliaria rubra undoubtedly caused by the irritating effect of the copious secretion on the skin." I feel sure that the vesicles of miliaria rubra in rheumatism are the later stage of sudamina. The contents of the vesicles in the clear stage give an acid reaction to litmus paper; after becoming milky they are usually, if not always, neutral or feebly alkaline.

Urine.—The elaborate researches which have been made into the condition of the urine in rheumatic fever have not thrown any light on the nature of the disease. The urine presents the same features as in other pyrexial conditions. Notwithstanding that patients are kept on a fluid diet, and that the thirst almost always present induces them to drink freely, the excessive loss of water by sweating usually reduces the quantity of urine passed below the normal amount. In colour it is a reddish yellow of varying intensity, and the specific gravity high (from 1020 to 1030); it is clear when first passed, and, unless the patient is under treatment, invariably deposits on cooling a heavy precipitate of pinkish or fawn-coloured urates; crystals of uric acid are not infrequent. The heightened colour of the urine depends on the presence of a large quantity of hæmatoporphyrin and a smaller quantity of urobilin. Dr. Garrod (38) failed to find any relation between the severity of the rheumatic attacks, or the destruction of blood corpuscles, and the amount of the colouring matter. The high specific gravity is due, not to an increase in the total amount of solids excreted, but to the diminished quantity of water passed. Senator states that the amount of urea passed exceeds the normal, notwithstanding that the diet may be very poor in albuminous substances. In cases treated by blistering fibrinuria has been observed.

Complications.[1]—Endocarditis, pericarditis, and, in less degree, myo-

[1] The association of palpitation and dilatation of the heart in young persons with rheumatism was (according to Dr. M. Baillie) first discovered in 1780 by Dr. David Pitcairn, physician to St. Bartholomew's Hospital (6).

carditis, can hardly be considered complications of acute rheumatism; they are just as much part of the disease as the affection of the joints. As we see certain joints escape, so in some cases we find no affection of the lining or investing membrane of the muscular tissue of the heart. The statistics of the frequency with which the different structures of the heart are involved in the disease are bewildering; partly from their numbers, partly from the different modes in which they have been drawn up, the majority of them having been collected to prove the advantage of some particular mode of treatment. I shall therefore confine myself to my own experience.

Endocarditis affects the sexes equally. It is difficult, perhaps impossible, to determine during an attack when endocarditis begins, for it gives rise to no special symptoms; but, in a large majority of cases, if no endocardial murmur be present during the first ten days of an attack the endocardium escapes.[1]

Pericarditis, on the other hand, occurs more frequently among men than women, and frequently begins at a much later period in the attack than endocarditis. It is usually stated that pericarditis is three times as frequent in men as in women; my own cases do not show nearly so large a preponderance of men. Out of 437 male patients pericarditis occurred in 65, and in 422 female cases in 42; that is to say, 14·87 per cent of the former, 9·95 per cent of the latter, and 12·45 for the whole number.[2] The risk of pericarditis is much greater in first attacks than in subsequent ones; out of 260 first attacks, 41, or 15·00 per cent, had pericarditis. The figures for first attacks show that age has nearly as important a bearing on the risk of pericarditis as it will be shown to have on endocarditis.

The onset of pericarditis is often attended by no special symptoms; neither pulse, respiration, nor general condition is altered. In other cases, before any pericardial rub can be detected, an increased frequency of pulse, a sense of oppression or pain in the cardiac region, increase in temperature, and an alteration in the sounds of the heart are to be observed. Clinically the important symptom is not so much the presence of pericardial friction as the extent of the præcordial dulness, which is a measure of the amount of effusion present. The effusion usually consists of organisable lymph, and it is often very large: fluid effusion is less frequent; when present it is generally serous, and rarely becomes purulent or blood-stained. In the seventy-nine cases of paracentesis pericardii collected by Dr. S. West (78), eleven, or possibly twelve, took place in the course of rheumatic fever. In the doubtful case pus was evacuated; in all the others the effusion was serum, or blood-stained serum; and, excepting in one instance where two pints were evacuated, the amount was small. The lymph is adherent to both the parietal and visceral layers of the

[1] On this point see also Gull and Sutton (43).

[2] The risk of drawing conclusions from statistics, unless the numbers are very large, is well shown in these figures, for the inclusion of my second series of cases, only 166 in number, raised the percentage in women more than 2 per cent.

pericardium; and its free surfaces, owing to the movements of the heart, become roughened in a peculiar manner, presenting a shaggy or honeycombed appearance closely resembling tripe. When much lymph is present it collects in large masses in the auriculo-ventricular groove and about the auricles. Pericarditis in cases terminating fatally is seldom unassociated with endocarditis. Clinical experience and the results of many post-mortem investigations show that it may be recovered from without leaving serious mischief; even when the surfaces of the pericardium become adherent little harm appears to result unless the adhesions be dense and thick, or the parietal portion of the pericardium is also adherent to the pleura or wall of the chest.

Dr. Cheadle has drawn attention to the grave results of pericarditis in children, from its interference with the growth and nutrition of the heart; and Dr. Sturges to the increased risks that are occasioned by its presence. As these conditions will be treated of in the article on "Acute Rheumatism in Children" I will not further allude to them.

The presence of *endocarditis* of greater or less severity is a very common accompaniment of the disease, and forms its most serious feature; as it leads to lasting and progressive degeneration of the cardiac valves. The endocardium over the mitral valve is especially prone to be attacked. Affections of the aortic valves alone are, comparatively speaking, rare; when they are involved, disease of the mitral valves also is almost always present (68). Endocarditis, either recent or old, was noted in about one-half of my own cases. The liability to endocarditis diminishes with the age. Dr. David Pitcairn's original observations on the connection between palpitation, dilatation of the heart, and rheumatism related to young persons; and Sir Thomas Watson says, "I have known only three persons pass through acute rheumatism with an untouched heart prior to the age of puberty, and in two of them I am by no means certain that the articular disease was true rheumatism." My own experience is very much the same. Among my hospital cases, in

36	under 10 years of age	29	had signs of old or recent endocarditis		= 80·55	per cent.
309	,, 20 ,,	223	,,	,,	= 72·16	,,
297	,, 30 ,,	159	,,	,,	= 53·53	,,
143	,, 40 ,,	58	,,	,,	= 40·55	,,
92	over 40 ,,	19	,,	,,	= 34·64	,,
12	age not stated	6	,,	,,	= 50·00	,,
889		494	,,	,,	= 57·50	,,

Taking first attacks, the percentage remains much the same during the first two decades, but decreases notably afterwards.

In 16	cases under	10 years of age endocarditis was present in			12 = 75·00	per cent.
,, 109	,, between 10 and 20	,,	,,	,,	60 = 54·13	,,
,, 75	,, ,, 20 ,, 30	,,	,,	,,	38 = 30·66	,,
,, 36	,, ,, 30 ,, 40	,,	,,	,,	13 = 33·33	,,
,, 8	,, over 40	,,	,,	,,	1 = 12·50	,,

In two of the four patients under ten, who are tabulated as escaping endocarditis, pericarditis occurred; but the sounds and size of the heart were normal on discharge from hospital.

The above figures show very strikingly that acute rheumatism is a disease of youth and early adult age, and that the younger the sufferers the greater the liability to cardiac implication.

Myocarditis.—It is probable that in all but the slightest cases of pericarditis a certain amount of myocarditis is present; in fatal cases, in which pericarditis is the cause of death, the outer layer of the muscular tissue of the heart is often found affected—either dark and infiltrated with leucocytes, or pale and of a yellowish hue, showing degenerative changes under the microscope; a similar but less marked condition is occasionally found in the muscular tissue subjacent to the endocardium. Dr. S. West (79) has drawn attention to *dilatation of the heart* occurring in rheumatic fever, or as an early sequel of it; and he has recorded a case in which well-marked symptoms of cardiac failure occurred without any evidence of endo- or pericarditis. A fatal case was met with, at St. Thomas's Hospital, in which acute granular degeneration of the muscular tissue was found after death; but in this instance pericarditis was present also. Somewhat similar cases have been recorded by Dr. Maclagan. Among my own cases I have met with a few in which an increased area of cardiac dulness, feeble first sound of the heart, and weakness of the pulse, have been present without any evidence of valvular or pericardial lesion; but how far the condition was due to an acute change in the heart's muscle I cannot venture to say. Rheumatic myocarditis, apart from peri- or endocarditis, I have never seen in the post-mortem room.

The clinical course and the effects of rheumatic inflammation of the heart and its coverings in young children differ in many respects from what we meet with in adults. The same may be said of rheumatic fever itself; for in young children the fever, as measured by the temperature, is seldom high, and the joint pains are slight. As acute rheumatism in young children will form a separate article in this work, the subject will not be further considered here. After the age of twelve or fourteen the symptoms present resemble those of adults.

The diagnosis of rheumatic fever, excepting in young children, seldom presents serious difficulty; nevertheless there are some conditions which may be mistaken for it, and occasionally it is impossible to form a differential diagnosis until the case has been under observation for some time. Osteo-arthritis in women who have been exhausted by child-bearing and lactation begins, I believe, much more frequently in an acute or subacute manner than is generally supposed. I have seen many cases beginning with slight fever and joint-pains resembling those of rheumatic fever, in which there was no tendency to cardiac affections, and no response to salicylate treatment; such patients after a time have slowly improved, but have been left with stiffness and some enlargement about the affected joints, and were destined, I fear, to become the victims of chronic osteo-

arthritis. Gonorrhœal rheumatism—or, more properly, synovitis—may be recognised by the presence or history of a urethral discharge, by the condition of the affected joints, and by the fixity of the pain in them.

Pyæmia, secondary to acute necrosis or osteomyelitis of some of the long bones—most commonly the tibia or fibula—or to disease of the petrous portion of the temporal, or less frequently to ulcerative endocarditis, which itself may be secondary to rheumatic affection of the valves, is the state most commonly confounded with acute rheumatism. The rarity of rigors in rheumatism, and their frequency and severity in pyæmia, may prevent this error: in acute necrosis of the long bones there are usually general swelling and tenderness of the limb affected, together with œdema; and in disease of the petrous bone either a history of a discharge from the ear, or the presence of it, together with tenderness over the mastoid process and some puffiness about the tissues in its neighbourhood. In pyæmia the joint or joints affected remain so, and the pain and swelling do not shift. The condition of the skin is also a guide—the skin in the pyæmic cases being often hot and burning, and rarely perspiring to the same degree as in rheumatic fever. When pyæmia is a part of ulcerative endocarditis the temperature has usually a decidedly hectic character; frequently there are tenderness and enlargement of the spleen due to infarction, and albumin and blood due to the same cause in the kidneys may be present in the urine.

A first attack of *gout* in a young subject may be another cause of hesitation for a time, but attention to the history and the absence of cardiac mischief will leave little doubt as to the diagnosis. In infants I have known *scurvy rickets* mistaken for rheumatism; in older children the swelling and tenderness accompanying hæmorrhage into a joint in *hæmophilia* simulate rheumatism very closely. The muscular and arthritic pains accompanying the early stages of *spinal disease* are often taken to be rheumatic, but they can hardly be confounded with rheumatic fever. Among the rarer conditions which should be mentioned is *glanders*, which at the outset is not infrequently mistaken for rheumatic fever.

Treatment.—While we remain as ignorant as we are at present of the immediate cause of acute rheumatism, its treatment remains in one sense empirical. It is profitless to go at length into the various methods of treatment which have been in vogue at different times since Sydenham first clearly defined the disease, and treated it (with the great exception of bleeding) on the principles by which it is treated now; nevertheless it will be advisable to review very briefly the methods that have been used. Sydenham (71) says, "No one doubts the inflammatory nature of pleurisy, and the blood of rheumatism is as like the blood of pleurisy as one egg is another. Hence the cure is to be sought in blood-letting." We can no longer uphold the father of modern clinical medicine in this view, but his rules as to regimen and diet still hold good. Nor should it be forgotten that he says (72), "With young persons and those who have not over-indulged in wine, rheumatism may be dispelled by spare and cooling diet, provided that it be moderately nourishing. This will

often do as well as repeated bleedings, which are but badly borne." Sydenham's teaching and practice held their ground until the middle of the present century, and it was chiefly due to the practice and teaching of Latham, Todd, Watson and Corrigan that the profession in this country recognised the error of venesection. Thus Latham says, "I have seldom employed venesection, and never largely": Todd says, "My experience leads me to value very lightly the efficacy of general bleeding in inflammation of the heart"; and Watson, "I seldom open a vein in these cases." The French school at the beginning of this century carried venesection to extreme excess, and somewhat later Bouillaud, to whom we owe much for his demonstrations of the connection between rheumatism and heart disease, was perhaps the greatest bleeder of them all. As bleeding fell into disuse purgation came in. Dr. Macleod (55), indeed, embraced both in his severe discipline; and Dr. Hope made use of calomel and opium together with salts and senna in doses sufficient to ensure four or five stools daily.

The alkaline system of treatment was brought into general prominence by Fuller (34), and was adopted by the bulk of practitioners in this country; though it never had the same popularity on the Continent. Founded on the theory that rheumatism was due to the presence of an acid in the blood, it was thought that if this acidity could be neutralised benefit to the patient would ensue.

The alkaline treatment still has strong supporters in this country, and I think it cannot be denied that it is beneficial; by depressing the heart's action it tends to lessen the danger of the occurrence of pericarditis, and possibly of endocarditis. Its greatest benefit was that its adoption led to the diminution or abandonment of the treatment by mercurials, which had been so universally inflicted on sufferers from endo- or pericarditis.

We now can ascribe little or no therapeutic value to the lemon juice treatment first introduced by Owen Rees (64), and used by Lebert and others abroad. Lebert made a very careful trial of it, giving the juice in increasing doses until six ounces a day were arrived at. It appears to me hardly necessary to do more than mention colchicum, veratria, aconite, tartar emetic, quinine, and other well-known drugs, which have each in their time been recommended on high authority; nor the more recently introduced antipyrin, phenacetin, and trimethylamine, which reduce the temperature indeed, but appear to have no really beneficial action on the disease. The marked anæmia which accompanies and follows acute rheumatism led to the administration of iron, especially the perchloride; but experience has shown that in the acute stage it is useless if not injurious.

The general experience that the fever and the pain in the joints are increased by movement, has led to the method of treating these cases by mechanical fixation of the joints with plaster of Paris or starch bandages; good results are said to have followed this plan, but I have no experience of it myself, and it has been little tried in this country. For a time the

treatment by blistering over or near the affected joints, so strongly advocated by Davies, was in favour; but no one, I think, would now maintain that it was of any assistance in the elimination of the morbid matter. Injection of various substances into the tissues surrounding the affected joints has been practised: Senator speaks favourably of his experience of Kunze's method (48), of injecting a 1 per cent solution of carbolic acid; and Dr. Gillespie has recorded some cases in which a 10 per cent solution was used with marked success so far as the relief of the pain was concerned.

Esmarch (25) and Stromeyer recommend the application of cold compresses or ice-bags, and many years ago Dr. Dovar says of rheumatism: "This is likewise a highly inflammatory fever; the blood does not appear more pleuritical or sizey in any distemper than this. Bleeding in this case is no remedy. I myself have known many lose a hundred ounces of blood and more without relief. The cure is much easier performed without that operation. Immersion in cold water is a remedy of singular use, as is evident from many hundreds that have been cured by cold water."

The use of willow bark in the treatment of intermittent and other fevers has been advocated, from time to time, for more than a hundred years (74); its virtues have been considered by some observers to be superior to those of cinchona. With the advance of chemical knowledge its active principle, salicin, was recommended for the same uses about sixty-five years ago (7), and about thirty years later salicylic acid was introduced to medical practice; at first mainly as a disinfectant. Almost simultaneously salicylic acid in Germany (65, 69) and salicin in this country were used for the treatment of acute rheumatism; and the results obtained were so favourable that the treatment of acute rheumatism by salicin, salicylic acid, or its compounds, quickly became general. The inconveniences in the use of salicylic acid were great; it is insoluble, disagreeable to take, and causes not infrequently gastric and intestinal irritation: it was soon found that its alkaline salts had the same beneficial effects without the disadvantages, and in this country salicylate of soda is used almost to the exclusion of other compounds. Salicin has not so great an influence over the disease, so far as my experience goes, as the soda salt; but it is useful in those cases in which the salicylate is ill tolerated.

I have seldom, if ever, seen serious or alarming symptoms produced by salicylate of soda, similar to those described by Dr. Greenhow, and alluded to by Sir William Broadbent and others, when due care was taken to diminish the quantity given as its effects are manifested. In cases of pericarditis, where myocarditis may be present without our knowledge, caution must be exercised in the use of salicylate, and the force and frequency of the pulse be noted at frequent intervals. Cerebral symptoms have been more frequently observed, not only the deafness, singing in the ears, buzzing and sound of rushing of water in the head which occur whenever the drug is pushed a little too far, but decided

wandering of the mind. This has occurred in my experience more frequently in women than in men, and has occasionally taken place before the patients appeared to be fully under the influence of the drug.

Experience shows that the most favourable treatment for cases of ordinary severity is as follows:—Twenty-grain doses of salicylate of soda every two or three hours until the patient is fully under the influence of the drug and the pain in the joints relieved, which usually happens in twenty-four hours or less; the drug should then be reduced in quantity, and as soon as the pain is gone and the temperature fallen to normal, the amount should be further reduced to half a drachm or a drachm in the twenty-four hours. This small dose should be continued for ten to fourteen days; indeed its continued administration appears to have a decidedly beneficial effect in warding off relapses.

If the tongue be coated and the bowels constipated, a free purgative of calomel, or calomel and colocynth, followed if necessary by sulphate of magnesia, is advisable; a free action of the bowels at the commencement of the salicylate treatment appears in great measure to obviate the unpleasant cerebral effects of the drug.

The diet should be fluid, and consist mainly of milk and farinaceous compounds, such as arrowroot, corn flour, rice, and so forth. A moderate amount of beef tea or other animal broths may be allowed. I have never seen advantage from rigidly cutting off all animal broths, as recommended by some writers (1). Plain water, barley water, lemonade, or imperial drink may be allowed at will to assuage the thirst from which the patients, notwithstanding their fluid diet, usually suffer. As a general rule, alcoholic stimulants are to be avoided; but in cases of prostration benefit is obtained from the administration of small quantities (not more than one to three ounces) of brandy in the twenty-four hours.

In cases complicated with severe endo- or pericarditis stimulants are more frequently required, and in them the diet must be more generous—eggs beaten up with milk and brandy, and a larger quantity of animal soups, being advisable.

Rest is most important, and for this purpose attention should be paid to the bed, which should be flat, smooth, and not too soft. Feather-beds are to be avoided, and the patient should be lightly clothed. Perspiration is so abundant that the sufferers are more comfortable when lying on a blanket than on linen, and they should wear flannel or merino bedgowns.

The existence of endo- or pericarditis calls for no special change of treatment. If in pericarditis much pain be present, a rare event, the application of a few leeches to the præcordium usually relieves it; where discomfort and oppression is felt rather than acute pain, a blister is of use; and blistering appears to favour absorption of the effusion. Dr. Lees speaks most favourably of the application of ice to the chest in pericarditis. In his experience, when applied continuously to the chest, it tends to check the violence of the local inflammation, hinders effusion, helps to cause absorption, and is usually grateful to the feelings of the

patient. I have no experience of its use in pericarditis; in croupous pneumonia it appears to me an uncertain mode of treatment; some patients like it and are relieved by it, but many are not able to tolerate it. No one now believes in the efficacy of mercury in controlling rheumatic pericarditis. It used to be given by the physicians of former generations in conjunction with opium; and I am convinced that in many cases in which dyspnœa and restlessness occur, marked benefit is obtained from the moderate and judicious administration of opium, which in these cases I prefer to morphia. Dr. Caton advocates the treatment of endocarditis by the application of a succession of small blisters to the cardiac region, and prolonged rest.

The pain and swelling around the joints are so capricious, and move so rapidly from joint to joint when no applications are made to them, that it appears doubtful whether topical applications have much effect either in relieving the pain or influencing the disease. Fomentations and all wet applications are troublesome and not to be recommended; belladonna or opium liniment applied on lint, covered with oil silk, sometimes appears of service, or wrapping the joints in simple cotton wool. It has lately been stated that oil of winter green, or salicylic acid, lanoline, oil of turpentine, of each one part, with lard, ten parts, applied externally to the joints act as well as when taken internally, being absorbed by the skin. In the few cases in which I have used this application, I have failed to find any evidence of absorption of salicylic acid as tested by its presence in the urine; and I have not seen any benefit to the patient. When tenderness and stiffness remain about a joint much relief may be obtained from properly directed passive movements and rubbing. Acute rheumatism rarely injures a joint permanently; and when ankylosis or persistent stiffness results, in all probability the original disease was not acute rheumatism.

The complications of acute rheumatism, other than those affecting the heart and hyperpyrexia, require but little comment. Pneumonia and pleurisy are the two most important concurrent conditions; and the former presents clinical features in rheumatic patients which differ from those ordinarily accompanying it. The amount of consolidation is often considerable, whilst cough is infrequent, and there is little or no expectoration. Pleurisy occasionally leads on to effusion, but among all my cases I have only once had to tap the chest. Pneumonia occurs more frequently than pleurisy, and is very generally an accompaniment of pericarditis. Statistics of the frequency of these complications are given by many observers, but differ widely. The proportionate numbers are much higher in the older tables than in the later ones, owing to the inclusion in them of pyæmic and septicæmic cases (37). Bronchitis, in anything like an acute form, has been rarer among my cases than pleurisy. Erythema multiforme (peliosis rheumatica) and nodosum, urticaria and tonsillitis occur in those subject to acute rheumatism, but rarely during the presence of an attack.

A consideration of the affinity between acute rheumatism and

chorea does not fall within the limits of this article. During the acute stage chorea is not common, but it is met with occasionally. Among the cases in which chorea has been present during the acute stage, several have been in young men beyond the age at which chorea is usually seen in the male sex. Two were under my own care, and the chorea, which was very severe, came on very shortly after pericarditis had set in. Conversely a girl was admitted into my ward with chorea, heart-murmur, and no joint affection, in whom hyperpyrexia supervened and ended fatally. After death a small patch of lymph was found on the surface of the left ventricle, and many vegetations on the margins of the mitral and aortic valves.

During the course of scarlet fever, and the period of convalescence from it, pain and swelling in the joints may arise. It seems probable that when synovitis occurs during the fever, it is pyæmic rather than rheumatic in character. Dr. Ashby, who has paid much attention to the connection between scarlet fever and heart disease, states that it occurs in children mostly from the seventh till the ninth day of the fever, and is usually met with in cases in which the temperature remains raised on account of severe inflammation of the cervical glands or ulceration of the tonsils and palate. The joint affection occasionally runs on to suppuration, and when occurring at this period is very rarely if ever accompanied by endocarditis, but is occasionally by pericarditis.

During desquamation and convalescence from scarlet fever, that is, after the first week or at a later period from the beginning of the fever, attacks of synovitis, which appear to be true rheumatism, are not very uncommon. They occur during convalescence from mild attacks quite as frequently as after severe ones; they are not infrequently accompanied by endocarditis, and occasionally by pericarditis. They are met with especially among young adults at the age at which rheumatism is most common, and in all other clinical features are indistinguishable from true rheumatism. Dr. Ashby is of opinion that scarlatinal nephritis is itself sufficient to set up endocarditis, and while questioning the conclusion that all post-scarlatinal endocarditis is necessarily rheumatic, admits that true rheumatism is not infrequent. [See article "Scarlet Fever," vol. ii. p. 122.]

Hyperpyrexia.—Since the regular use of the clinical thermometer the nature of the cases of rheumatic fever characterised by marked and decided cerebral symptoms has become known. These cases were formerly thought to be due to a metastasis of the rheumatic inflammation from the joints and heart to the meninges of the brain, and were spoken of as cerebral rheumatism. Senator says, "Acute cerebral or spinal meningitis may set in," and quotes two cases, one from Lebert and one from Dr. Stretch Dowse (22), in support of this statement. I have referred to the case reported by the latter, and find no reason for thinking it was rheumatic. Dr. Dowse considered it to be a case of acute arachnitis in a dissolute woman exposed to great cold and hardship immediately before the beginning of her fatal illness, and he does not attribute it in

any way to rheumatism. The majority of the cases formerly regarded as rheumatic meningitis were undoubtedly pyæmia; yet meningitis apparently does occur, as in the Clinical Society's report on hyperpyrexia two instances are said to have been met with among twenty-four post-mortem examinations after death from hyperpyrexia.

The cases in which hyperpyrexia supervenes do not as a rule differ at the outset from others; many of them are described as being mild, until the alarming symptoms arose. In not a few of the fully reported cases the sufferers are stated to have been in a more or less depressed mental condition for some time prior to the advent of the rheumatic attack; and several authorities have expressed an opinion that these cases are met with more frequently in patients whose constitutions have been weakened by alcoholic or other excesses; but there appears to be no good evidence in support of this view.

Hyperpyrexia is said to occur more frequently among men than women. Taking the cases collected by the late Dr. Wilson Fox (31), the Committee of the Clinical Society, and myself, we have 107—sixty-two occurring in the male, and forty-five in the female sex.

It is impossible to say what proportion cases of hyperpyrexia bear to the number of attacks at different ages; but its occurrence in early childhood is as yet unknown: the earliest age at which it has been noted was in a boy of twelve (16). Of the 107 collected cases—

16	occurred in	persons under			20	years of age.
48	„	between	20	and	30	„
25	„	„	30	„	40	„
11	„	„	40	„	50	„
5	„	„	50	„	60	„
2	the age was not stated.					
107						

From these figures it would appear that no period of life, except early childhood, is exempt from this complication; after the age of sixty rheumatic fever is of extreme rarity. Hyperpyrexia is met with more frequently in first attacks than in subsequent ones. Of the 107, fifty-seven were in the course of a first, twenty of a second, eight of a third, two of a fourth or subsequent attack; whilst in twenty the number of the attack is not recorded. The mortality also appears greater in first attacks than in subsequent ones.

What elevation of temperature is to be called hyperpyrexia? The Clinical Society's Committee divided the cases into three groups: those of undoubted hyperpyrexia, with temperatures of 106° F. or upwards; those showing a marked tendency to a continued and persistent temperature of a high range (104° F.); and a third group (under which only three cases are included), in which the symptoms usually accompanying hyperpyrexia were present but without an excessive temperature. I think cases

not exceeding 105° F. should hardly be considered as cases of hyperpyrexia; and in my eighteen cases I have not included any which did not exceed this standard. The danger to life is, generally speaking, in proportion to the height of the temperature, but recovery has taken place after the temperature has reached 111° F. (27).

With the rise of temperature the commonest concurrent symptoms are diminution of the pain in the joints and sometimes complete cessation of them; extreme restlessness passing into acute delirium, or drowsiness deepening later into stupor: spasm of certain groups of muscles, or general convulsions are not uncommon. The profuse sweating diminishes, and in some instances perspiration appears wholly suppressed, and the skin is intensely hot and burning to the touch. With the rise in temperature we find great acceleration of the pulse and rate of breathing. In some patients a large increase in the amount of urine passed has been noted, and not infrequently persistent and uncontrollable diarrhœa comes on. Death is almost always preceded by coma.

Although, with the advent of hyperpyrexia, it is undoubtedly true that in many cases the articular pain diminishes, or at all events is less complained of, the Clinical Society's Committee found that in many the condition of the joints remained unaltered, and that pericarditis was present in more than half; 70 per cent of those who recovered had pericarditis at the onset of hyperpyrexia, and in 42 per cent of the fatal cases the same condition was found after death.

The rise of temperature occupies a variable amount of time. The highest point may be reached within a few hours of the commencement of the symptoms; or the maximum may not be met with until several days of high temperature have passed (36). In cases which have not been treated by direct application of cold, the highest temperature is usually at the time of death; and in several instances it has been observed to rise for a short time after death. A continuously elevated temperature, especially if it occur in a patient whose joint pains have moderated, and in whom there is no evidence of pericarditis or other complication, should give warning of the possibility of hyperpyrexia; and means should be taken to reduce the temperature. The probability of its occurrence is still further increased if, in addition to a continuously high temperature, unusual restlessness or vigilance be present.

Hyperpyrexia is most apt to arise during the second week of the fever, but instances have been known as early as the second day and as late as the twenty-ninth (17). Out of seventy-four cases in which the date of its occurrence and of the beginning of the attack was known, hyperpyrexic symptoms commenced on the seventh, eighth, or ninth day in 25 (33·7 per cent).

Causation of hyperpyrexia.—The group of symptoms of which rapid rise in temperature to an extreme point is the most remarkable feature is not confined to rheumatism. A similar condition is met with in small-pox, scarlatina, typhus and enteric fevers, pneumonia, and more frequently, perhaps, in pyæmia; yet there can be no doubt that this variation arises

much more frequently in acute rheumatism than in any other disease. Rheumatic hyperpyrexia appears to be more prevalent in some years than in others, so that cases are apt to occur, as it were, in runs; but there does not appear to be any direct connection between the prevalence of rheumatic fever and the number of hyperpyrexic cases. Hyperpyrexia occurs, in London at all events, with much greater frequency in the summer months—June, July, and August (17)—in which the total amount of rheumatism is small as compared with the other portion of the year.

At present the *pathology of hyperpyrexia* is unknown. The heat-regulating mechanism of the body is in some way or other thrown out of gear, and the cerebral symptoms are probably dependent on the circulation through the brain of unduly hot blood (59, 76), producing symptoms similar to those met with in abnormally high temperatures of other specific diseases and of insolation (see vol. i. p. 491). Dr. Andrew (1) remarked many years ago that the suddenness of the rise of temperature is remarkably similar to what constantly occurs at the close of cerebral disease in its coarser and more common forms,—hæmorrhage, softening, meningitis, or cerebral tumour. In what way the heat-regulating centres (if they exist) are primarily affected is unknown. Lebert was in favour of the view that in acute disease, under certain circumstances, a poisonous material is formed in the body which causes paralysis of the nervous centres; others have supported the view that the increased heat due to febrile metabolism of tissue is sufficient of itself to cause paralysis of the nervous centres on which the heat-regulating mechanism depends. In whatsoever way the paralysis of the heat-regulating centres be produced, a vicious circle is set up, resulting in an overheated condition of the body and a consequent deterioration of tissue, the muscular tissue of the heart especially suffering.

Treatment of hyperpyrexia.—Experience has shown that in these cases, as in insolation, no antipyretic drugs are of any use. The only successful mode of treatment is the rapid abstraction of heat, and by this means we are able to snatch some sufferers from the very jaws of death. Dr. Meding appears to have been the first to save life in this way—in the case of a woman in whom hyperpyrexia occurred about the twentieth day of an attack of rheumatic fever following erysipelas. The rheumatic attack was of moderate severity and complicated with endocarditis; the temperature rose to 108·6° F. In the same year Dr. Wilson Fox (32) published his first case treated unsuccessfully by repeated immersion in cold and tepid baths, and the following year his two successful ones (33). Although the value of cold affusion in the treatment of fevers had been recognised for years, and its efficacy in insolation had been proved by Dr. Morehead and others of large Indian experience, it was not until after the publication of Dr. Wilson Fox's cases that the treatment of hyperpyrexia by the rapid abstraction of heat was systematically used in this country. Prolonged experience has proved its efficacy not only in "rheumatic," but also in other forms of

pyrexia; and many lives have been saved by this means. When this mode of treatment was first made use of, the patients were placed in baths of 90° F. or higher, and the water gradually cooled. Experience has shown that nothing was gained by this, the temperature of the bath need not be raised above 65° F.—the more rapidly heat can be abstracted from the patient the better, and the less the subsequent exhaustion; hence it is a good plan to add ice rather than water of the temperature of the air to the bath as it is heated by the patient. Cold-water packing alone is not of much effect; but ice packing and rubbing the body with lumps of ice have secured results nearly as satisfactory as the cold bath, and can be applied when facilities for bathing are not at hand. Care should be taken to sponge the head with ice-cold water or to apply an ice-cap whilst the patient is in the bath. The duration of the bath depends on the rapidity of the fall in temperature and the condition of the patient. It is desirable, if possible, to keep the patient in the bath until the temperature has fallen five or six degrees. If decided shivering comes on, the patient must be taken out of the bath, even if the temperature has not fallen many degrees; not infrequently the temperature continues to fall for a time after removal from the bath. To combat the collapse which sometimes follows, the free use of stimulants is required; and should the temperature sink below the normal, and blueness and lividity of the face or extremities remain, hot bottles should be applied to the feet and back. Abstraction of heat being the only method as yet known of benefiting these patients, the cold bath must be repeated as often as necessity arises; and patients have recovered after undergoing twenty-six baths; in one case during thirteen, in another during sixteen days (16).

The presence of pneumonia or of peri- or endocarditis, though probably adding to the danger of a fatal issue, does not in any way contra-indicate the use of the cold bath.

It is difficult to form an estimate of the proportionate number of cases of hyperpyrexia which are saved by this treatment, as the fatal cases are not reported so freely as those in which recovery takes place; we have no data, therefore, for estimating the frequency of the occurrence of hyperpyrexia. The mortality of the collected cases to which reference has been made was as follows:—

	Cases.	Deaths.	Recoveries
Clinical Society's Report	67	33	34
Dr. Wilson Fox's cases	22	19	3
Other cases collected by the Author	18	5	13
	107	57	50

Omitting 18 cases of Dr. Wilson Fox's collected cases, in which the

application of cold was not tried, there remain 89—39 deaths and 50 recoveries.

Sequels.—Besides those pertaining to the heart and its coverings the sequels of the disease are few. It has already been remarked that it is rare for a joint to suffer permanent injury; in this respect the disease offers a strong contrast to osteoarthritis, gonorrhœal rheumatism, and gout. The most serious evil, other than cardiac mischief, which it leaves behind it is the tendency to further attacks and *anæmia*. Dr. Garrod has extended and in the main confirmed the observation of Hayem on the condition of the blood in acute rheumatism; he shows that an attack is always accompanied by a very considerable fall in the number of the red corpuscles, and that this fall takes place with great rapidity; besides the diminution of the red, both observers are agreed that there is a slight increase in the number of the white corpuscles. Dr. Garrod further remarks that in cases running a favourable course without serious pulmonary or cardiac lesions, the red corpuscles very rapidly rise to the normal standard again. Hayem states that in protracted cases the red corpuscles fall in numbers continuously, but Garrod found that having fallen to a low level the numbers of the red corpuscles remained stationary. The variations in the worth of the red corpuscles, by which Garrod means their proportion of hæmoglobin, follow closely the variations in their number; but in some instances the percentage of hæmoglobin in the corpuscles continues to fall during convalescence, although the number of red corpuscles may increase. He sums up his conclusions thus (38): "The anæmia of rheumatism is of two kinds: (1st) an acute oligocythæmia developing during the acute stage, and rapidly recovered from as soon as convalescence sets in; and (2nd) a pseudo-chlorotic condition which is developed as a sequel of the attack in a few cases, and which unless it is appropriately treated may last for a long period, the fall of worth continuing in spite of the amelioration of the patient's condition in other respects."

The appropriate treatment of these anæmic patients is the main difficulty in the management of convalescence from rheumatic fever; in other cases, simple attention to the digestion and regulation of the bowels is all that is necessary, and bark, the old remedy for acute rheumatism,[1] often appears to me of use and preferable to quinine. Anæmic patients, and those in whom there has been endo- or pericarditis, should not be allowed to get up until they have made very considerable progress. Iron is frequently ill tolerated in the anæmic condition immediately consequent on an attack, although at a later period it may be of great benefit; cautious trial may be made of the various preparations of iron, as, if well borne, they usually relieve the anæmia quickly: in patients who are unable to take ferruginous tonics, arsenic is of use, and I have not infrequently seen great benefit from cod-liver oil.

The gravity of the *cardiac lesions* left by endocarditis frequently does not show itself until long after convalescence has been established;

[1] Used by Haygarth on the recommendation of Dr. Fothergill.

it is essential, therefore, that all violent exercise or efforts calculated to strain the heart should be avoided. At the same time, sufficient exercise to improve the general health and to keep up the vigorous nutrition of the heart is of the utmost importance.

Dilatation of the stomach is not a very rare sequel of acute rheumatism, and is a grave obstacle to recovery [*vide* art. "Dilatation of the Stomach."]

In connection with rheumatic fever mention must be made of the *rheumatic nodules* — small, firm, subcutaneous, fibrous nodules — which are found with much greater frequency in children than in women or men. In children they are of considerable pathological and clinical importance, as severe cardiac disease may accompany them without well-marked joint pains. Dr. Cheadle says (13) "they are found most commonly upon the back of the elbow, over the malleoli, and at the margin of the patella. They are also found not infrequently upon the head, especially along the superior curved line of the occiput, the temporal ridge, and now and again on the extensor and flexor surfaces of the hands, or the extensor of the feet, the vertebral spine, the spine of the scapula, and the crista ilii." They vary greatly in size, and, not giving rise to pain, are often overlooked; when of large size, however, they become painful from pressure, and the larger ones often remain for months. Dr. Cheadle considers that the eruption of large nodules signifies persistent and uncontrollable cardiac disease, which almost invariably goes on to a fatal termination. [See Fig. 1, p. 46, art. "Acute Rheumatism of Childhood."]

Their histological structure is of great interest, for they present microscopically a structure very similar to that found in the nodular growths on the cardiac valves, if not identical with them; the rapidity and frequency with which these subcutaneous nodules disappear render it probable that in fortunate cases the similar growths on the cardiac valves may likewise disappear without leaving mischief behind. There is no doubt that subcutaneous nodules are most commonly associated with disease of the cardiac valves. In the cases collected by Drs. Barlow and Warner (4), twenty-seven in number, there was but one in which cardiac disease was not well marked; nevertheless, several cases have been reported in women (12, 79, 23) in whom no morbid sound at the heart was noted, and in adults their pathological meaning and bearing on prognosis do not appear to have much value.

The appendices need a few words of explanation and comment.

The Army Medical Reports do not give the returns for rheumatic fever in the United Kingdom, all cases being entered under rheumatism: on applying to the Medical Department of the Army, the Director-General kindly furnished me with the figures for the years 1889-91, which are here made use of.

Egypt, Canada, the Cape and St. Helena stations are entered twice over, the entries in italics being for longer, the others for shorter periods; the high average of admissions to hospital in Egypt and the

Cape for the longer periods are due to the years during which our troops were on active service in the field. In Egypt the average admission during the years of active service reached the high figure of 29·68 per thousand; in the Cape, 6·48 per thousand. I have consequently taken the average of the years during which our troops were not on active duty in the field as the normal.

The Canadian returns were unsatisfactory; in 1882 the military returns do not give the number of cases, but say of rheumatism—"various forms rather prevalent." In 1883 the admissions to hospital for rheumatism amounted to 57·70 per thousand, and the note appended is, "but the acute form was not as numerous as in the preceding year." In the next two years rheumatic fever is separated from rheumatism, and the figures for these years give the very high average of 21·67 per thousand, followed in 1886 by a drop to 2·33 per thousand. I have thought it best, therefore, to consider the average of the six years 1886-91 as more truly representing the normal. It is possible, of course, that a long epidemic of rheumatic fever might have been going on during the years 1882-85; but I failed to obtain any returns of the civil population during those years for comparison.

Dr. Newsholme, in the Milroy Lectures of the year 1895, when dealing with the returns of the Army Medical Department, did not separate rheumatism from rheumatic fever. A comparison of Appendix I. and Appendix II. shows how necessary it is to do this if we are to form an opinion as to the prevalence of acute rheumatism at any station: for example, he states, correctly enough, that, among our Mediterranean stations, Gibraltar is the worst station for rheumatism, and Cyprus the best; although the admissions to hospital for rheumatism are nearly twice as many per thousand at Gibraltar as at Cyprus, for acute rheumatism they show exactly the same percentage. Similarly, the Cape of Good Hope, which in Appendix II. heads the list for our white troops, is one of the freest from rheumatic fever. Mauritius, which stands first in Appendix I., is twelfth in Appendix II. England, Ireland, and Scotland occupy much the same position in both lists, but there is a much greater difference in acute rheumatism than in rheumatism; in England, the average number of attacks of acute rheumatism per thousand is double that in Scotland, whereas in rheumatism it is only 7 per cent higher. Of all our military stations Bermuda is the freest both from rheumatism and rheumatic fever; the latter fact is remarkable, as the mortality from rheumatism among the civil population is high, averaging—for the five years 1889-93—5·53 per thousand of deaths. It is interesting also to note that, whilst so-called rheumatism is extremely prevalent among our coloured troops at Sierra Leone, rheumatic fever is almost unknown; yet the same troops in the West Indies have a higher percentage for rheumatic fever than our white troops quartered there.

Turning to the Colonial statistics (Appendices III. and IV.), I regret that those of rheumatic fever are so scanty. Appendix III., with the exception of Mauritius, Jamaica and Ceylon, is confined to people of

European, and for the most part British blood; whereas Appendix IV. includes in many cases an enormous preponderance of native and coloured blood.

The effect of malaria in producing pains and aches classified as rheumatism is well shown in Appendix IV., Honduras standing first Australia and New Zealand cannot be considered malarious countries and I was surprised to find them stand so high in both III. and IV Unfortunately no available statistics could be obtained for New South Wales and Queensland.

Appendix V. is inserted for what it may be worth. I think no conclusions can be drawn from it as to the frequency of rheumatic fever among the general population; nor as to its prevalence in different parts of the country: for instance, in two districts so near together as Manchester and Salford, the latter stands at the head of the list with 10·24 rheumatic fever cases to every 100 medical ones; and yet at the Royal Infirmary, Manchester, the percentage is only 3·34. If we take the proportion of acute rheumatism to the other medical patients in the five London hospitals given in Appendix VI. as a fair test of the frequency of the disease in London, we see that it is about 6½ per cent; this percentage is equalled or exceeded by ten hospitals in the list, and they seem to have little in common. Salford, Barnsley, Bristol and Leeds may be considered as manufacturing communities with a large labouring population. An admixture of gout may account for the high figures at Burton-on-Trent; but these explanations do not hold good for Whitehaven, Tewkesbury, Oxford, Brighton, and Hastings, which have few features in common, whether industrial or climatic (*vide* vol. i. p. 50).

The east coast appears to suffer decidedly less than the west; Newcastle-on-Tyne, which is a vast manufacturing city, is alone in presenting a high average.

In compiling this list, care has been taken to make use only of the returns of those hospitals which afforded full information as to the actual numbers of their rheumatic fever or acute rheumatism cases; and of the annual number of their medical as distinguished from their surgical patients.

I am glad of this opportunity of tendering my thanks to the secretaries and resident officers who have so kindly supplied me with the information which enabled me to compile Appendix V., at a trouble and labour to themselves which I fear was considerable.

[TABLE

APPENDIX I.—Abstracted from the Army Medical Reports, showing the average Number of Cases of Rheumatic Fever per thousand of strength admitted into Hospital at the various stations.

Stations.	Admissions to Hospital per 1000 of strength.	Remarks.
Egypt	7·02	10 years, which include the years of active service.
Canada	6·26	8 years.
1. Mauritius	3·75	10 years.
2. Malta	3·71	10 years.
3. England	3·43	3 years, 1889-91.
Cape and St. Helena	3·12	9 years, includes the years of active service.
4. Canada	2·83	6 years, 1886-92.
5. India	2·50	10 years.
6. Gibraltar	2·38	10 years.
Cyprus	2·38	10 years.
8. Ireland	2·03	3 years, 1889-91.
9. China and Straits Settlements	1·94	10 years.
10. West Indies Coloured Troops	1·75	4 years.
11. Egypt	1·73	5 years, subsequent to the conclusion of active service.
12. Scotland	1·52	3 years, 1889-91.
13. Cape and St. Helena	1·30	5 years, subsequent to the conclusion of active service.
14. West Indies	0·83	10 years.
Ceylon	0·83	8 years.
16. West Africa Coloured Troops	0·70	10 years.
17. Bermuda	0·42	5 years.
18. Native Army of India	0·29	3 years, 1889-91.

APPENDIX II.—Abstracted from the Army Medical Reports, showing the average Number of Cases of Rheumatism per thousand of strength admitted to Hospital at the various stations of the British Army during the years 1882 to 1891.

Stations.	Admissions for Rheumatism per 1000 of strength.	Remarks.
1. West Africa Coloured Troops	84·74	10 years.
2. West India ,, ,,	63·15	4 years.
3. Cape of Good Hope and St. Helena	42·94	9 years, includes the years of active service.
4. Gibraltar	35·18	10 years.
5. West Indies	33·42	10 years.
6. India	32·68	10 years.
7. Canada	31·09	6 years, 1886-92.
8. England	31·06	3 years, 1889-91.
9. Malta	29·91	10 years.

APPENDIX II. (*continued*)

Stations.	Admissions for Rheumatism per 1000 of strength.	Remarks.
10. Ceylon	28·13	8 years.
11. Ireland	27·90	3 years, 1889-91.
12. Mauritius	26·86	10 years.
13. Egypt	26·79	5 years, subsequent to the conclusion of active service.
14. Scotland	24·20	3 years, 1889-91.
15. Native Army of India	20·87	3 years, 1889-91.
16. Cyprus	19·04	10 years.
17. China and Straits Settlements	17·09	10 years.
18. Bermuda	16·18	5 years.

APPENDIX III.—Abstracted from the Blue Books and Official Returns of our Colonies and Colonial Dependencies, showing the average Number of Cases of Rheumatic Fever admitted to the Civil Hospitals per thousand of admissions from all causes.

Stations.	Admissions for Rheumatic Fever per thousand of Total Admissions.	Remarks.[1]
Province of Ontario	22·07[2]	The figures for rheumatism being 19·74.
Victoria (Australia)	13·71	6 years.
New Zealand	13·55	6 years.
Victoria Hospital	13·45	Halifax.
Western Australia	7·08	1 year only.
South Australia	6·66	
Cape of Good Hope	5·78	
Tasmania	5·05	This high average is due to the prevalence of the disease in 1891.
St. Helena	4·96	
Mauritius	4·44	
Jamaica	2·02	6 years.
Malta	0·68	
Ceylon	0·18	

[1] The average of the five last years for which statistics have been obtainable has been taken.

[2] Does the high figure for Acute Rheumatism depend on the nature of the classification?

APPENDIX IV.—Statistics of Rheumatism abstracted from the Official Returns of our Colonies and Colonial Dependencies, showing the average Number of Cases of Rheumatism admitted to the Civil Hospitals per thousand of admissions from all causes.

Stations.	Admissions for Rheumatism per thousand of Total Admissions.	Remarks.[1]
1. Honduras	92·07	
2. Victoria (Australia)	71·32	6 years.
3. Jamaica	56·11	6 years.
4. Straits Settlements	53·95	4 years.
5. Sierra Leone	53·56	
6. New Zealand	53·33	7 years.
7. Nova Scotia	44·25	
8. Western Australia	42·71	
9. Province of Ontario	41·82	
10. Cape of Good Hope	41·10	
11. South Australia	38·44	Adelaide Hospital only.
12. Ceylon	36·30	
13. St. Helena	30·44	
14. British Guiana	28·40	
15. Victoria Hospital (Halifax)	26·45	
16. Mauritius	23·79	
17. Tasmania	23·79	
18. Malta	22·71	

[1] The averages are taken from the last five years for which I could obtain statistics, except when otherwise stated.

[TABLE

APPENDIX V.—Showing the Proportion of Acute Rheumatism to the Total Admissions for Medical Diseases in various Provincial Hospitals in Great Britain and Ireland.

Hospitals.	Rheumatic Fever. Per cent.		Remarks.
1. Salford	10·24	5 years	
2. Whitehaven	9·53	10 ,,	
3. Burton-on-Trent	9·26	10 ,,	
4. Barnaley	8·07	7 ,,	Victoria Hospital. Acute and subacute.
5. Tewkesbury	7·66	10 ,,	
6. Oxford	7·64	11 ,,	
7. Bristol	7·20	10 ,,	Royal Infirmary.
8. Brighton	6·74	10 ,,	
9. Leeds	6·59	7 ,,	
10. Hastings	6·56	5 ,,	
11. Sheffield	6·06	11 ,,	Approximate only. One-half of cases being stated to be acute.
12. Kilmarnock	5·75	10 ,,	
13. Birmingham	5·57	10 ,,	General Hospital.
14. Douglas, Isle of Man	5·33	9 ,,	
15. Bedford	5·14	11 ,,	
16. Dorchester	5·13	10 ,,	
17. Glasgow	5·10	10 ,,	
Newcastle-on-Tyne	5·10	5 ,,	
Stoke-upon-Trent	5·10	5 ,,	
20. Newark	4·70	10 ,,	
21. Salisbury	4·68	10 ,,	
22. Derby	4·60	6 ,,	
23. Birmingham	4·28	11 ,,	Queen's Hospital.
24. Guildford	4·17	11 ,,	
West Norfolk and King's Lynn	4·17	6 ,,	
26. Hereford	4·04	10 ,,	
27. Liverpool	3·96	10 ,,	Royal Southern Hospital.
28. St. Patrick Duns	3·77	8 ,,	Dublin.
29. Exeter	3·76	3 ,,	
30. Folkestone	3·73	5 ,,	
31. West Meath and Mullingar	3·57	5 ,,	
32. Manchester	3·34	12 ,,	Royal Infirmary.
33. Dundee	3·30	8 ,,	
34. Aberdeen	2·81	8 ,,	
35. Guernsey	2·74	7 ,,	Cottage Hospital.
36. Leamington	2·64	5 ,,	Warnford Hospital.
37. Perth	2·53	3 ,,	This is calculated from an average of medical cases.
38. Kent and Canterbury	2·52	10 ,,	
39. Edinburgh	2·35	1 year	One year only.
40. Chichester	1·83	10 years	
41. East Suffolk and Ipswich	1·41	10 ,,	
42. Louth Dispensary	0·97	8 ,,	
From the following hospitals, the returns did not permit the cases of rheumatic fever to be separated from the rest of the rheumatic cases.			
43. Torbay	12·35	9 years	All cases said to be acute.
44. Gravesend	5·96	10 ,,	
45. Liverpool	5·56	8 ,,	Majority acute. Royal General Infirmary.
46. Huntingdon	5·00	...	An average only.
47. Grantham	3·59	10 ,,	
48. Huddersfield	2·00	10 ,,	An average only.

APPENDIX VI.—Showing the average Proportions of Cases of Rheumatic Fever admitted to some of the London Hospitals to the Total Admissions to the Medical Wards.

Hospitals.	Percentage of cases of Rheumatic Fever.	Remarks.
Westminster Hospital . . .	7·37	9 years from 1884-92.
St. George's Hospital . . .	6·60	10 „ 1884-93.
University College Hospital . .	5·74	10 „ 1881-90.
St. Thomas's Hospital . . .	6·13	10 „ 1882-91.
St. Bartholomew's Hospital . .	6·63	21 „ 1873-93.
If the last five years of the series is taken, a very great diminution in the number of cases at the Westminster, St. Thomas's, and St. Bartholomew's Hospitals is shown. University College Hospital shows a fractional decrease, and St. George's a fractional increase.		
Westminster Hospital . . .	3·70	5 years from 1888-92.
St. Thomas's Hospital . . .	4·78	5 „ 1887-91.
St. Bartholomew's Hospital . .	3·51	5 „ 1889-93.
University College Hospital . .	5·22	5 „ 1886-90.
St. George's Hospital . . .	6·22	5 „ 1889-93.

W. S. CHURCH.

REFERENCES

1. ANDREW. *St. Bartholomew's Hospital Reports*, vol. x. pp. 356, 359.—2. ASHBY. *British Med. Journal*, Sept. 15, 1883, p. 514; *Lancet*, May 22, 1886, p. 961.—3. AUSTEN, J. A. *Lancet*, July 7, 1883.—4. BARLOW and WARNER. *Transactions of the International Medical Congress*, 1881, vol. iv. p. 116.—5. BABES, Prof. "Address delivered before a General Meeting of the Eleventh International Medical Congress, Rome, 1894." Translation, *British Medical Journal*, 1894, vol. ii.—6. BAILLIE, M. *Lectures and Observations on Medicine*, 1825, p. 184.—7. BLAINCOURT, I. B. *Essai sur le salicine, etc.* Paris, 1830.—8. BOWLBY, A. *Injuries and Diseases of Nerves*, 1889, p. 52.—9. BROADBENT, Sir WM. *Presidential Address Clinical Society*, vol. xx. p. lxxii.—10. CANSTATT. *Specielle Pathologie und Therapie*, 1854, vol. i. p. 604.—11. CATON, R. "Treatment of Endocarditis," *Lancet*, Aug. 17, 1895.—12. CAVAFY and MONEY, A. *Pathological Society's Transactions*, vol. xxxiv. pp. 43, 49.—13. CHEADLE. Harveian Lecture, *Lancet*, April 27, 1889, p. 825.—14. COATS, J. *Journal of Pathology*, vol. i. No. 2, p. 216.—15. Collective Investigation Inquiry, *Brit. Med. Journal*, 1888, p. 387.—16. *Clinical Society's Transactions*, vol. xv. 1882.—17. *Clinical Society's Report*, vol. xv. p. 264.—18. DALTON. *Brit. Med. Journal*, March 1, 1890, p. 472.—19. DAVIES, H. *On the Treatment of Rheumatic Fever in its Acute Stages exclusively by Free Blistering.* London, 1864.—20. DAY, W. H. *Medical Times and Gazette*, 1867, vol. ii. p. 225.—21. DOVAR. *A Physician's Legacy to his Country*, 1733.—22. DOWSE, S. *Lancet*, July 6, 1872.—23. DUCKWORTH, DYCE. *Clinical Society's Transactions*, vol. xvi. p. 53.—24. EDLEFSEN. *Zur Statistik und Aetiologie des acuten Gelenkrheumatismus.*—25. ESMARCH. *Berliner klin. Wochenschr.* 1871, p. 423.—26. FAGGE and PYE-SMITH. *Principles and Practice of Medicine*, 3rd edition, vol. ii. pp. 688, 706.—27. FENNY. *Lancet*, July 4, 1885, p. 17.—28. FIESSINGER. *Gazette médicale de Paris*, No. 14, p. 160, April 1890.—29. FOSTER, MICHAEL.

Text-Book of Physiology, Part ii. p. 695, 5th edition.—30. FOSTER, W. B. *British Medical Journal*, 1871, vol. ii. p. 720.—31. FOX, WILSON. *On the Treatment of Hyperpyrexia*, 1871.—32. *Idem. Lancet*, July 32, 1870.—33. *Idem. Lancet*, Aug. 12, 1871, p. 213.—34. FULLER, W. H. *Rheumatism, Rheumatic Gout, and Sciatica*, 3rd edition, p. 19.—35. GABBETT, H. S. *Lancet*, Oct. 20, 1883.—36. GARLAND. *Lancet*, September 6, 1884, p. 409.—37. GARROD, A. E. *A Treatise on Rheumatism*, pp. 39, 52, 53, 106.—38. *Idem. Roy. Med. and Chir. Soc. Trans.* vol. lxxv. pp. 194, 203, 206 (1892), and *Journal of Pathology and Bacteriology*, vol. i. No. 2, p. 193, Oct. 1892. —39. *Idem. Journal of Physiol.* vol. xvii. 1894, p. 349.—40. GILLESPIE. *Medical Press and Circular*, June 17, 1891.—41. GOODHART, J. F. *Clinical Society's Transactions*, 1880, vol. xiii. p. 123.—42. GREENHOW. *Clinical Society's Transactions*, vol. xiii. 1880, p. 327.—43. GULL and SUTTON. *Royal Med. and Chir. Soc. Trans.* vol. lii. p. 70 *et seq.* 1869, and vol. liii. p. 43.—44. HAIG. Wood's *Medical and Surgical Monographs*, New York, Feb. 1890. *Trans. R. Med. and Chir. Society*, vol. lxxiii. 1890.—45. HAYEM. *Du sang et de ses altérations anatomiques*. Paris, 1889.—46. HIRSCH. *Handbook of Geographical and Historical Pathology*, vol. iii. pp. 754, 765. Syd. Soc. Transl. —47. KULZ. *Beiträge Pathologie und Therapie des Diabetes Mellitus*, B. ii. 1872.—48. KUNZE. *Deutsche Zeitschrift f. pract. Medizin*, 1874, No. 10.—49. *Lancet*, Aug. 12, 1871, p. 213.—50. LATHAM, P. W. *Croonian Lectures*, 1886.—51. LEES, D. B. *Brit. Med. Journal*, Feb. 18, 1893; and *Lancet*, July 22, 1893.—52. LONGSTAFF, G. B. *Studies in Statistics*, 1891, pp. 313, 319.—53. LYONS. *Report on the Pathology of Diseases of the Army in the East*, 1856, p. 88.—54. MACLAGAN, T. J. *Clinical Society's Transactions*, vol. xxvii. p. 182.—55. MACLEOD. *On Rheumatism and on the Affection of Internal Organs, more especially the Heart and Brain*. London, 1842.—56. MANTLE. *British Medical Journal*, 1887, vol. i. p. 1383.—57. MEDING. *Archiv für Heilkunde*, 1870, p. 467.—58. MITCHELL, J. K. *American Journal of Medical Science*, 1831, viii. p. 55.—59. MURCHISON. *Lancet*, May 21, 1870.—60. NEWSHOLME. Milroy Lectures, *Lancet*, March 16, 1895, p. 660.—61. POORE, V. *Lancet*, 1868, June 13, p. 751.—62. RATHERY. *Gazette des hôpitaux*, 1869, May 20.—63. RAYHER. *Virchow's Archiv*, 1861, xxi. p. 85.—64. REES, OWEN. *On the Treatment of Rheumatic Diseases by Lemon Juice*, 1849.—65. REISS. *Berliner klin. Wochenschrift*, 1875, p. 691.—66. RICHARDSON, B. W. *The Cause of the Coagulation of the Blood, etc.*, 1858, p. 371.—67. SENATOR. Ziemssen's *Cyclopædia of the Practice of Medicine*, English Trans. vol. xvi. pp. 15, 17, 18, 41, 42, 44.—68. SIBSON. Reynolds' *System of Medicine*, vol. iv. p. 494.—69. STRICKER. *Berliner klin. Wochenschrift*, 1876, pp. 1-15.—70. STURGES, O. Lumleian Lectures, *Lancet*, 1894.—71. SYDENHAM. Medical Observations. *Sydenham Soc. Trans.* vol. i. pp. 254, 257.—72. *Idem. Processus Integri*, chap. x. section 9.—73. TODD. *Croonian Lectures*, 1843, p. 143.—74. WARING. *Bibliotheca Therapeutica*, Sydenham Society, 1878.—75. WATSON, Sir THOS. *Lectures on the Principles and Practice of Physic*, vol. ii. p. 312, 4th edition.—76. WEBER, H. *Clinical Society's Transactions*, vol. i. p. 21.—77. WEBER, L. *New York Medical Record*, Aug. 31, 1889, p. 238.—78. WEST, S. *Roy. Med. and Chir. Soc. Trans.* vol. lxvi. 1883, p. 259. —79. *Idem. St. Bartholomew's Hospital Reports*, vol. xiv. p. 228; vol. xvi. p. 197; vol. xxii. p. 215.—80. WUNDERLICH. "On Temperature in Disease," *Sydenham Society's Trans.* p. 396.

W. S. C.

The Acute Rheumatism of Childhood

Acute rheumatism, as it occurs in childhood, presents many points of difference from the disease as it appears in maturer life. The ordinary conception of acute rheumatism, as characterised by swollen, tender, painful joints, profuse sour-smelling perspiration, and high pyrexia, is drawn from observation of this affection as we see it in adults; in them arthritis is one of the most constant, prominent, and characteristic of the morbid phenomena. This description, however, does not represent rheumatism as it is usually seen in childhood; at this period of life it has a far wider pathological range, and its phenomena are more various and comprehensive.

The Rheumatic series.—Certain affections of other parts and organs are so frequently associated with acute rheumatism of the joints that the existence of some close pathological connection between them is now very generally accepted; and these affections appear more frequently and play a more prominent part in the disease as it is presented in childhood. Endocarditis and pericarditis, for example, which are frequent accompaniments of articular rheumatism in adults, appear yet more frequently and in more intimate association with the rheumatism of childhood.

In addition to these there are other manifestations which appear so commonly or exclusively in association with rheumatism in the case of children that they must be admitted into the rheumatic series; namely, subcutaneous tendinous nodules, exudative erythema, and chorea: not to mention a few rarer conditions of less certain connection. In the rheumatism of children these non-arthritic manifestations become conspicuous and frequent; while articular inflammation, so prominent in the rheumatism of adults, is usually slight and unimportant, and occasionally may be absent altogether. Tonsillitis occurs in connection with the rheumatic state in children, as in adults; but has no special features or significance.

In the case of children, then, arthritis is not the eminent or representative symptom. It would seem that at this period the joint tissues are less susceptible, the other fibrous tissues, the skin, and the nervous system more so than in later life. Thus in the rheumatism of childhood arthritis is at its minimum; endocarditis, pericarditis, subcutaneous nodules, chorea are at their maximum. As life advances this rule is gradually reversed: the joint affection grows more prominent, regular, and characteristic, while the other phenomena decline and tend to die out. Endocarditis and pericarditis become less frequent; subcutaneous nodules, so significant in early life, practically cease with the advent of puberty; and chorea, so common in connection with

the rheumatism of childhood, disappears as maturity is reached. If the picture of acute rheumatism had been drawn originally from the disease as it appears in childhood, when it arises under simpler conditions, the articular affection would not have been regarded as representative; endocarditis and pericarditis, and possibly chorea, would have been looked upon as the primary and essential phenomena, and arthritis as a complication.

Another general point of distinction between the rheumatism of childhood and that of later life is the tendency of the various phases to arise independently and apart from each other. They do indeed occur together—arthritis, for example, with pericarditis and endocarditis, or endocarditis with chorea and nodules; or all these together as phenomena of the same rheumatic storm: yet the series of rheumatic events is often spread out or scattered over a term of months or years, so that the history of a rheumatism may be the history of a whole childhood. At one time, for instance, there may be an endocarditis, a chorea at another, and an arthritis at another, without any further coincident manifestation of the disease. Again, the rheumatic series, as seen in children, may be complete or incomplete in any degree. The whole series of phenomena may follow in succession, or the expression of the rheumatic attack may be limited to a single event. An arthritis, an endocarditis, or a chorea may occur alone, without the subsequent manifestation of any other rheumatic seizure. Again, the combinations of the several phases may follow any order of sequence.

Sometimes—perhaps most frequently—an arthritis appears first; in other cases an endocarditis; now and again a chorea inaugurates the morbid series. These phases may, moreover, be closely associated in point of time; or may occur singly, or again in groups with varying intervals between.

Influence of sex.—The incidence of acute rheumatism upon the sexes in childhood differs remarkably from that in the period of maturity. Taking all ages of life together it falls nearly equally on males and females, somewhat more heavily on the former. In childhood, however, the case is reversed, and the disease is most common in girls. The proportions, however, show curious variations.

According to the statistics yielded by the reports of the Collective Investigation Committee, in the first period of from 1 to 5 years of age boys preponderate in the proportion of 5 to 1. In the next quinquennial period, from 6 to 10 years, they become nearly equal, in the proportion of 15 to 14. In the next period of 11 to 15 years there comes a remarkable change: the proportion is suddenly reversed, the girls suffering in the proportion of nearly 2 to 1. This explains in some degree, probably, the greater prevalence of chorea in girls about this period of life. After the age of 15 the greater liability of girls declines up to 20 years, so that at the close of this period men preponderate.

Another point with regard to acute rheumatism, which comes out

with especial clearness and force in the case of children, is the potent influence of hereditary disposition. The statistical evidence recorded in this respect is unsatisfactory and, indeed, obviously defective. The results show great discrepancy, owing to differences in the thoroughness of the inquiry, the nature of the evidence allowed, and the class of patient from whom it is drawn. Most of the material was obtained from hospital patients of the poorer class, who generally know little of their relations, or of the diseases from which they suffer; and the inquiry was usually based upon the history of articular affection alone. More trustworthy data can be obtained from private cases; parents of the better class know a good deal about the diseases of their own children and of immediate blood relations. Statistics from private cases of acute rheumatism under my own observation give a proportion of 70 per cent in which there is a definite family history of the same affection in near blood relations. Many examples have been recorded which show a remarkable family incidence, the individual cases often occurring apart, and not, therefore, attributable to locality or infection. The most significant fact of all is the power which double inheritance appears to exercise in intensifying the tendency to the disease. When acute rheumatism prevails in the families of both parents, not only is the liability to the disease increased, but its severity and persistence are increased also. The most inveterate and fatal cases which have come under my observation have had this double strain; and similar examples have been recorded by Dr. Goodhart, Dr. Garrod, and others.

As the phenomena of acute rheumatism in childhood are more varied and extensive than those met with in later life, so each in turn presents points of difference from the same condition in adults, and requires detailed and individual consideration.

Arthritis.—It has already been stated that in childhood the articular affection is usually slight, indeed it is less extreme in every way than in the case of adults; there is less swelling and tenderness, less pain and fever. It is a comparatively rare thing to see a young child lying motionless, bound hand and foot, and not daring to move or turn in bed for fear of pain—a condition so characteristic of the disease in older people. There is often merely a little pain, stiffness and tenderness, without perceptible swelling, limited perhaps to a single joint or set of joints. Sometimes the affection of the fibrous tissues is extra-articular, confined to the tendons of the fascia in the neighbourhood of the joint, and analogous to the stiff neck, which sometimes indeed is the only sign of rheumatism. The tendons of the hamstring muscles under the knee, for example, may be attacked alone, causing stiffness and pain on movement; a child thus affected walks on the tips of the toes, with knees bent to avoid tension of the sinews—a very characteristic sign. Such minor attacks are apt to be overlooked altogether, or regarded as simple feverish attacks, not severe enough to require medical aid, and soon forgotten. Yet, slight as the articular signs may be, they are frequently accompanied by a subacute endocarditis or pericarditis, leading

eventually to grave heart lesions. These facts afford the key to many instances of unexplained heart disease and of chorea.

The slight arthritis of acute rheumatism in childhood often assumes a misleading aspect, so that it may be difficult to distinguish it from other ailments involving pain and tenderness of limbs and joints. Its early recognition is, however, of the first importance, since, as previously stated, an insidious and deadly endocarditis or pericarditis may be at work concurrently with it. The affection of the hamstring muscles, with walking on the toes, and apparent inability to put down the heel, has been mistaken for incipient talipes, and treated accordingly. Similarly, the disinclination to walk has been attributed to paresis and loss of power to do so. Conversely, other morbid conditions are often mistaken for articular rheumatism. In my experience the mistake is most frequently made in infantile scurvy. The swelling, immobility, and extreme tenderness of the limbs, accompanied often by slight transient pyrexia, at first sight simulate articular rheumatism very closely. Scurvy, however, may usually be distinguished by the periosteal position of the swelling, which, moreover, is almost invariably limited to the shafts of the long bones. In one of my own cases there was periosteal swelling over the malar bone; and in another a swelling of the periosteum of the lower part of the shaft appeared to extend to the joint structures. Such cases, however, are rare exceptions. Other diagnostic points are the existence of spongy gums, subcutaneous and other hæmorrhages, hæmaturia or albuminuria; although these are not always present. Moreover, infantile scurvy is limited to the bottle-feeding period of infancy, that is, to the first eighteen months or two years of life, when articular rheumatism is almost unknown.

Infantile palsy (essential paralysis, acute anterior polio-myelitis) in its early stage, when there is often considerable hyperæsthesia, is another affection liable to be confounded with acute rheumatism. The chief points of distinction are that in infantile paralysis there is extreme flaccidity of muscle; the affected limbs are limp and fall loosely down with drooping of the toes, while the tenderness is found to be diffused and general, not confined to joints and tendons. Later the disappearance of faradic contractility and tendon reflex is decisive. Another condition liable to be mistaken for slight rheumatic arthritis is syphilitic disease of the ends of the long bones. In this there is tenderness and swelling from accompanying periostitis, and there may even be some articular inflammation, with pain on movement. It is to be distinguished from true rheumatic affection by the presence of other signs of congenital syphilis, or by the history; but the most distinctive feature is the age at which it occurs, namely, in the first few months after birth, when rheumatism is practically unknown. I have never met with a case of acute rheumatism in early infancy, although rare instances have been recorded by Senator, Henoch, and others. Possibly these may have been examples of syphilitic affection, or of infantile scurvy, a disease at that time hardly recognised. The swelling of the wrists in tetany, the arthritic hæmorrhages of

hæmophilia, the joint trouble of tubercular disease, the arthritis of pyæmia, and acute osteomyelitis, are also conditions met with in children which are liable to be mistaken for slight rheumatic arthritis.

The inflammation of the joint structures in children being slight, certain symptoms especially associated with it are slight also. The profuse acid perspirations, so marked in the acute rheumatism of grown persons, are not common in children. Pyrexia, again, is rarely great, and this is the more remarkable as in children the temperature tends to rise from slight causes. In rheumatism, so eminently pyrexial a disease as far as adults are concerned, the temperature in the case of children seldom rises above 101° or 102°; degrees of 103°, 104° are comparatively uncommon, and usually of short duration.

Hyperpyrexia, which occurs now and again in the acute rheumatism of adults and is so grave and often uncontrollable, is at least extremely rare in childhood. I have never seen an instance of excessive or fatal hyperpyrexia in a child, nor any case in which the temperature has shown a persistent tendency to run up rapidly beyond control to a fatal height. The earliest age I can find recorded at which fatal hyperpyrexia occurred is thirteen.

Anæmia.—The effect of acute rheumatism in producing anæmia is well known, and in children this is even more noticeable than in adults. When the rheumatic state is actively manifested in children anæmia proceeds apace, and hæmic cardiac murmurs are frequent. With the single exception of that of diphtheria there is no organic poison which causes such rapid blanching as that of rheumatism.

Cardiac inflammations.—In children, as Dr. Sturges pointed out, more than in adults the heart is apt to suffer in all its structures at the same time: endocarditis, pericarditis, and myocarditis are liable to occur together, and the carditis to be general. This, however, chiefly arises towards the close of fatal cases, when rheumatic attacks recur, and endocarditis has already given rise to valvular lesions. Then pericarditis, accompanied by myocarditis, is usually added to the original endocardial inflammation. As a rule, however, endocarditis occurs first and alone.

Endocarditis occurs acutely in the course of articular rheumatism in children as it does in adults. More often it comes on in a subacute, insidious, progressive form, which is characteristic of the disease in childhood, when the arthritis may be slight, or at the time even inappreciable. Endocarditis may indeed appear at any stage of the rheumatic procession of events, early or late, entirely alone or in combination with arthritis or chorea, or nodules, or pericarditis, or with any or all of these combined. Usually it comes early in the series and recurs, wherein it stands in contrast with pericarditis which is apt to appear towards the close. Most often it arises in connection with slight arthritis or with chorea; and, not seldom, valvular lesion, due to antecedent endocarditis, is discovered already established when the first joint affections or chorea attract attention and lead to examination of the condition of the heart.

As might be expected from the fact that in children endocarditis arises in connection with slight manifestations of the other forms of acute rheumatism, or even apart from them, inflammation of the endocardial valve structures is nearly twice as common in the case of children as in that of adults.

It has been stated that endocarditis is liable to come on insidiously, without its existence being known or suspected until some other rheumatic manifestation leads to examination of the heart, and to the discovery of valvular mischief already established. Even when it arises in the course of an attack of articular rheumatism, and is discovered at the outset, the signs of it are vague, slight and slowly developed; some quickening of the pulse, with an excited, uneven, irregular action of the heart, and changes in the cardiac sounds afford the earliest indications. The most common of the latter is the soft, blowing, systolic murmur at the apex with accentuation of the pulmonary second sound, which indicates a commencing mitral leakage, due to the thickening and rigidity of the flaps and chordæ which lead to imperfect closure of the valve.

Another early sign is reduplication of the second sound, not at the base but at the apex; it is probably due to a retarded opening of the mitral gate resulting from rigidity caused by inflammatory thickening. With this there are sometimes—not usually at the outset but after a time—a soft, blowing, diastolic murmur after the later of the two second sounds, and an accentuation of the first sound, signs of changes which gradually lead to stenosis and, long afterwards, to the development of a true presystolic rumble. Although, as a rule, endocarditis in children sets in and progresses insidiously and slowly, giving little outward sign of its existence, and often recurring frequently in subacute form, occasionally it proceeds with great rapidity. In such cases there is more fever and constitutional disturbance, and the valvular inflammation progresses quickly; so that a murmur which began as a soft, gentle, blowing sound, the very existence of which appeared doubtful when first detected, may (according to my observation) increase to a loud musical bruit in the course of a single week. The concurrent eruption of subcutaneous nodules is always a grave sign indicative of serious and generally persistent or recurrent valvulitis, often accompanied by pericarditis also. Rheumatic endocarditis in children, as in adults, may attack either the mitral, aortic, or tricuspid valves: yet the first is by far the most common event, and mitral regurgitation the most usual lesion; nevertheless mitral stenosis is more common than is generally believed. Mitral stenosis, indeed, is the special product of the subacute, slow recurrent course which is so characteristic of endocarditis in childhood, and it is in the time of childhood that the mischief usually begins. The reduplication of the second sound at the apex, or the slight accentuation of the first, which are the early signs of it, escape notice. These signs are the results of the stiffening of the valve-flaps from inflammatory thickening, which produces virtual stenosis; although there is no fixed

constriction of the orifice. The loud, vibrating, prolonged, presystolic bruit and slapping first sound are not common in childhood; they do not usually appear until some years later, when the disease is advanced, and the mitral orifice has become permanently narrowed. Hence the rarity of a pronounced presystolic bruit in early childhood, and its discovery with such startling frequency about the age of puberty. Thus also, no doubt, arose the error of regarding mitral stenosis as usually non-rheumatic in origin.

Ulcerative endocarditis is a most rare sequel to the rheumatism of children, since it is most often met with in subjects broken down by drink and disease. The only case within my own observation occurred in a girl of eight years old, and this is the sole instance which I can find recorded in a patient under the age of fourteen.

Pericarditis may arise at any step in the rheumatic series; first or last; alone, or combined with any one or more of the other manifestations, such as endocarditis, arthritis, the evolution of nodules, or chorea. Most often, however, it comes late, in association with recurrent endocarditis, when the heart is already hypertrophied and dilated. So that, as Dr. Sturges pointed out, it is almost always found post-mortem in the fatal heart disease of children. As in the case of endocarditis, pericarditis in children is usually subacute, chronic, recurrent. It may occur acutely, as in the rheumatism of adults, with rapid exudation and effusion of fluid, pericardial distension and distress, but this course is exceptional; as a rule it proceeds insidiously, with slight symptoms, although these are more distinct and recognisable than those of valvular inflammation.

The first appreciable physical sign of subacute pericarditis is usually a slight pericardial rub, which may be constant, or intermittent, or may cease altogether. The child is seen to be restless and uncomfortable; and, if old enough, complains of pain in the præcordial region, which is generally a little tender on firm pressure. The pulse quickens to 120 or 130, out of proportion to the temperature, which is usually but slightly raised, say to 99° or 100° or 101° F. This quickened pulse-rate, without adequate rise of temperature, is very characteristic of the subacute pericarditis of early life. Anæmia increases in a marked degree; chorea if present becomes aggravated, or curious emotional attacks come on, in which the child is moved to tears or laughter by a word. With these signs a mitral murmur perhaps appears, or an existing murmur grows louder and rougher; subcutaneous nodules also often arise on the elbows and knees, or on the ankles or occiput; these are always suggestive of a grave implication of the fibrous structures of the heart. The pericardial rub, if it has ceased, reappears after an interval; or the extent of cardiac dulness may show the variations due to a fluctuating amount of effusion; the effusion, however, is never large, and usually is quickly reabsorbed.

Further—the most significant indication of all—although the physical signs of friction and effusion subside, due probably to the formation of

pericardial adhesions, the rapid action of the heart, in spite of digitalis, belladonna, ergot, or strophanthus, continues unchanged. The area of cardiac dulness increases, and there is muffling of the sounds over the mid-cardiac region—probably not due to effusion, but to the increased size of the heart and thickening of the pericardium. Enfeeblement, anæmia, emaciation proceed apace; the pulse grows weaker; and so after weeks or months the patient sinks from heart failure and exhaustion, without dropsy or marked dyspnœa: or the symptoms may subside to recur at intervals, and the course of the disease thus extend over years instead of months.

Occasionally endocarditis and pericarditis, together with myocarditis, run an acute course; delirium supervenes, respiratory distress becomes great; vomiting, which is often obstinate and persistent, not infrequently sets in, and prostration becomes extreme. Such symptoms always indicate a condition of great danger. These cases as a rule arise when pericarditis occurs late, when the heart is already seriously damaged by previous attacks of endo- or pericarditis, and the secondary changes of dilatation and hypertrophy, and perhaps adherent pericardium, have already advanced to a marked extent.

Pericardial fibrosis.—The results of the chronic recurrent pericarditis of childhood are serious enough. In many instances the pericardium becomes greatly thickened, and its two surfaces so closely adherent that, when examined post-mortem, all sign of the space between them is found to have disappeared, and the heart is enclosed in a tightly-fitting sac of fibrous tissue, sometimes as much as an eighth of an inch in thickness. The chronic inflammatory process spreads now and again from the pericardial sac to the tissues of the pleura and adjacent mediastinum, so that these become matted together in a thick fibrous mass—an indurative mediastino-pericarditis. This is indicated clinically by ascending dulness along the middle and upper sternum, and increased respiratory and cardiac distress.

In certain cases fibrous growths cause pressure upon the great vessels at the base of the heart: such pressure is most operative upon the veins, leading to hepatic engorgement, cirrhosis with ascites, imperfect filling of the pulmonary arteries, cyanosis, dyspnœa and general dropsy.

These cases of mediastino-pericardial fibrosis are rare; but several have come under my own observation, and others have been recorded.

Another occasional result of this chronic fibrous pericarditis of childhood is worthy of notice. The tight, strangling grip of the inelastic fibrous sac not only interferes with the contraction and dilatation of the cardiac chambers, and thus causes grave embarrassment of the heart's mechanism, but it interferes also with the progressive development of the organ in childhood, so that it fails to grow in proportion to the rest of the body, and general dropsy eventually follows as the result of imperfect cardiac power.

Cases of the kind are recorded by Watson and Bouillaud, and several have come under my own observation.

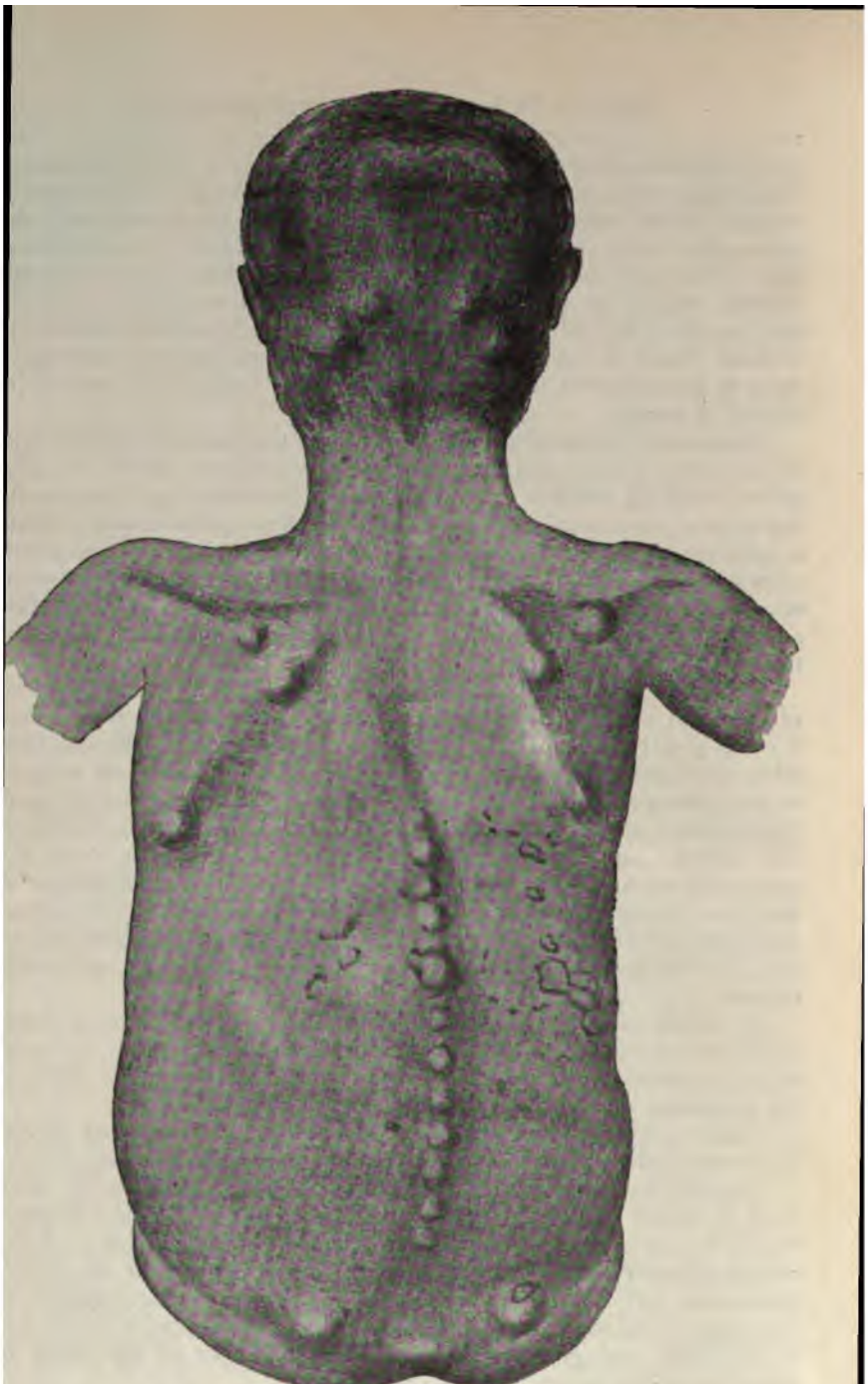


FIG. 1.—Acute rheumatism. Grave form, with numerous large nodules. **Fatal.**

7. S., æt. 4 years and 3 months. (Hospital for Sick Children, Great Ormond Street. December 10, 1887. Under the care of Dr. Cheadle.) Rheumatic nodules, erythema marginatum, chorea, double mitral murmur, arthritis.

Myocarditis.—With pericarditis, and probably to some extent with endocarditis, there is always some degree of inflammation of the muscle lying beneath; or rather perhaps of the intermuscular connective tissue, extending to the muscle fibres themselves. These are found after death to be swollen, blurred in outline, with numerous nuclei, and proliferation of the interstitial fibrous tissue.

In old-standing cases of chronic pericarditis, with adherent pericardium, there is marked increase of interstitial fibrosis, and the striæ of the muscle fibres become indistinct or are altogether lost. These changes of interstitial fibrosis and degeneration of muscle fibre account in part for the cardiac failure which is so conspicuous in the last stage of heart disease in children.

Hypertrophy and Dilatation.—All that need be said about these secondary changes is that, other things being equal, these changes proceed more rapidly in the case of children than in adults. In accordance with the general law that in early life the tissues respond more quickly and readily to stimulation, and grow more vigorously than after maturity, hypertrophy develops faster, and dilatation is more rapid; probably because the tissues are softer and more yielding.

Dropsy and other Complications.—The ready development of hypertrophy, in response to increased strain, renders compensation exceptionally complete at first, and probably accounts for another special feature of the rheumatic heart affections of children, namely, that the common complications of disease of the heart in adults, such as pulmonary apoplexy, enlargement of the liver and extreme dropsy, are rare in the younger children. It is an unusual thing to see a little child blue, turgid, and waterlogged—a sight so sadly frequent in the last stage of valvular disease in adults. In addition to effectiveness of compensation, however, there is another reason for this immunity: children with severe heart disease usually die from other causes before the stage of grave tricuspid leakage is reached—from anæmia, wasting, asthenia, rather than from direct obstruction to the mechanism of the circulation; unless it be in the case of the strangling grip of pericardial fibrosis and adhesion.

Subcutaneous tendinous nodules.—Another exceptional and special feature of acute rheumatism in children is the development of subcutaneous nodular growths of fibrous tissue. Not uncommon in early life, they are comparatively rare phenomena in the case of adults. They may appear in connection with any of the other phases of rheumatism, but are mostly associated with endocarditis and pericarditis, and in this relation are of serious import. They appear to have been first noted by Dr. Hillier, and subsequently by Meynet and others; but their comparative frequency and clinical significance were first pointed out by Dr. Barlow and Dr. Warner. The nodules lie under the skin in connection with fascia or tendons, that is, in relation with fibrous tissue; and similar nodular proliferations have been found in the periosteum, and even in the pericardium. They vary in size from that of a hemp seed to that of an almond, or are even larger; when small they may not become visible until the

skin is drawn tightly over them, although easily detected by the touch. They are not tender except slightly so in rare instances; nor is there redness of skin over them unless it arise casually from friction or pressure. There may be but one of these nodules; but three or four are usually

FIG. 2.—Section of subcutaneous tendinous nodule in acute rheumatism, showing active proliferation and cell infiltration of fibrous tissue.

John T., æt. 7½ years. (Hospital for Sick Children, Great Ormond Street. About August 6, 1888.) Chorea, arthritis, endocarditis, pleurisy, nodules, pericarditis.

found; sometimes indeed the number is large, as many as twenty or thirty. In one extreme instance of an intense rheumatic state within my observation, it was calculated that 200 were present at one time. These fibrous nodules grow to a perceptible size in the course of a few days; but, when large, they take a considerable time to attain to their

full dimensions. Their duration varies from a few days to several months —the minimum period recorded being three days, and the maximum five months. When examined microscopically, they are seen to consist of nuclear growth in all stages of transformation into fibrous tissue—a pro-

FIG. 3.—Section of aortic valve in ulcerative endocarditis in a child, showing proliferation and cell infiltration of subendothelial fibrous tissue, similar to that of the subcutaneous nodule shown in Fig. 2.

liferation analogous to that which is seen in the interstitial framework of the liver, or of the kidney, in cirrhosis. The connection of these nodules with rheumatism and rheumatoid arthritis is apparently absolute. So far as is known they own no other origin or relation.

General experience has confirmed views originally expressed by Dr. Barlow and Dr. Warner as to the grave significance of these fibrous out-

LANE LIBRARY

growths. They indicate concurrent and usually progressive cardiac disease; that a similar process of inflammatory change and proliferation is going on in the fibrous tissues of the cardiac valves or pericardium, or in both. When the nodules are many and large they indicate serious danger, an endo- or pericarditis which is uncontrollable, and advances almost certainly to a fatal ending. In the acute rheumatism of children especially they have this ominous association with progressive endocarditis and pericarditis of the most serious kind. [See, however, pp. 91, 92.—Ed.]

Microscopical examination of a section of the mitral valve in rheumatic endocarditis shows proliferation and cell infiltration of the subendothelial fibrous tissue, which is the counterpart of that which takes place in the evolution of a subcutaneous nodule. This may explain the clinical significance of the latter; while their rapid development and equally quick disappearance in some cases suggest that the concurrent mitral murmurs, which are apt to arise and disappear, are not functional, but are due to the development of similar inflammatory cell growth in valves and chordæ, which may subside on occasion as quickly as we see it does sometimes in a subcutaneous nodule.

Eruptions associated with acute rheumatism.—*The exudative erythemas and purpura rheumatica* are far more frequently associated with the rheumatic state in the case of children than of adults. They may appear at any stage of the rheumatic series, but generally arise in connection with active disturbance of other kinds, such as arthritis; and not infrequently with the endocarditis and pericarditis of the more serious cases.

The erythemas occur chiefly in children, in marginate, papular, or urticarious forms; erythema nodosum is less often concurrently associated with other phases of rheumatism than the rest. Its appearance, however, from time to time in immediate connection with a rheumatic outburst seems to stamp it as one of the rheumatic phenomena, and it is occasionally met with in childhood.

Purpura rheumatica is an eruption or hæmorrhagic condition which occurs almost exclusively in childhood. Some cases of rheumatic purpura are clearly extreme instances of erythema, in which hæmorrhagic extravasations are unusually marked. Others, again, are possibly instances of simple purpura or hæmophilia, in which there is oftentimes tenderness and swelling of adjacent joints. There is, however, a purpura closely associated with acute rheumatism, occurring concurrently with general arthritis, or in subjects who have suffered from articular rheumatism. In the latter case it has an arthritis of its own, the joints in proximity to the eruption — especially the feet and ankles when it appears in the legs —being swollen, tender, and painful, although there may be no rise of temperature. In a few rare instances this condition is accompanied by hæmaturia.

Tonsillitis is another rheumatic phase which is perhaps somewhat more common in children than in adults; but it shows no special features in early life.

Pleurisy, pneumonia, bronchitis, and other inflammatory conditions of minor importance occur occasionally in the course of rheumatism in childhood as in later life, but they have no distinctive characters at the early period which call for remark.

Chorea.—One of the most interesting properties of acute rheumatism in childhood, and one almost entirely confined to that period of life, is its connection with chorea. Genuine chorea, apart from mere grimacing (which is a disturbance of the second dentition, and not in any way connected with rheumatism), is very closely associated with the rheumatic state. It appears not only in direct connection with acute rheumatism of the joints, but with endocarditis also, with pericarditis, with erythema multiforme and nodosum, and, above all, with that especial rheumatic sign the evolution of subcutaneous nodules. Chorea may appear in relation to one or more of these; and when it occurs alone, apart from any other rheumatic manifestations at the moment, is often followed at an interval by arthritis, or by other rheumatic manifestations. It is especially liable to occur, even when alone, in members of families in which rheumatism is rife; one individual may have articular rheumatism, another endocarditis, another chorea. The chain of evidence in favour of the rheumatic nature of chorea in the case of children is remarkable. It is impossible to state it in detail here, but it may be affirmed that the presumption is strongly in favour of the view that, in the great majority of cases at all events, chorea is of rheumatic origin. We may almost certainly say that the endocarditis and pericarditis of chorea are invariably rheumatic in nature and origin. [*Vide* art. "Chorea" in a later volume.]

In relation to this may be mentioned the curious association of the neurotic groundwork of chorea with the rheumatic taint. According to my own observation, the rheumatic child in a family is the nervous child of the family; but no distinct association with specific nervous family disease can be satisfactorily traced.

Scarlatinal rheumatism.—The articular affection which appears now and again in the course of scarlatina, and which can in no way be distinguished from that of acute rheumatism, is naturally a form met with chiefly in childhood. It is often accompanied by endocarditis or pericarditis, sometimes by chorea; and subcutaneous nodules have more than once been observed in the course of an attack.

This scarlatinal rheumatism usually arises early; it has been observed on the third day, but occurs most frequently about the end of the first week. And it is noteworthy that the especial liability to acute articular rheumatism which obtains in girls extends also to the scarlatinal form. [*Vide* art. on "Scarlet Fever."]

Rheumatoid arthritis occurs occasionally in childhood as a sequel of acute rheumatism, sometimes in its most severe and intractable form. Such cases are, however, rare, and present no distinctive characters. [*Vide* p. 102.]

Prognosis.—The prognosis of acute rheumatism is far more serious in the case of children than in that of adults. This results chiefly from

the greater tendency to endocarditis and pericarditis, the proclivity to which may be roughly stated to be inversely as the age of the patient. The danger from this source is no doubt aggravated by the fact that in many cases the first stages of heart disorder are apt to be overlooked, owing in some cases to the slight character of the articular affection which accompanies it, and to its entire absence in others. Thus the cardiac inflammation runs on unchecked by the palliation of rest and appropriate treatment. A further source of increased peril in childhood lies in the remarkable tendency of the disease at this period to recur, so that the heart is liable to become fatally injured by repeated attacks of endocarditis and pericarditis.

In mild cases, no doubt, the rapid growth of muscle favours quick and complete compensation; but in severe cases the ready dilatation of the young tissue more than counterbalances the rapidity of its remedial growth.

Treatment.—The affection of the joints, with its accompanying pain and tenderness, being in the case of children almost invariably of small intensity, and not the cause of any great distress, the pyrexia also being in most instances moderate—all symptoms, in fact, both articular and constitutional, being subdued and subacute—anything like active treatment by full doses of salicylate of soda, so effective in the case of adults, is rarely required. Not infrequently, indeed, it does great harm by its depressing effect, for children bear severe and drastic measures badly. In place of salicylate of soda, salicin may be given in doses of from 5 to 20 grains, or quinine in doses of 1 to 3 grains, every four to six hours. With this an alkali should be combined, such as citrate of soda or of potash, in doses of 5 to 20 grains according to age.

The general evidence of statistics of the Collective Investigation Committee is in favour of the conclusion of the late Dr. Fuller and of Dr. Dickinson, that cardiac inflammation is less frequent and less pronounced under full alkaline treatment than under any other. In the case of children this is especially important, for the chief interest and danger centres in the cardiac implication and its results, and not upon the pain and distress of articular troubles. The one great object of treatment should be to diminish this danger as far as possible.

The first and most important point is to guard against an insidious or masked attack of endocarditis or pericarditis. It has been shown that this may accompany the slightest articular affection, or be associated with chorea, or an exudative erythema, or an eruption of subcutaneous nodules; or again it may arise apart from any known rheumatic phase with an indeterminate febrile attack of apparent insignificance. It is essential, therefore, to examine the condition of the heart carefully, not only in every case where there is the slightest articular or other known rheumatic condition, such as a stiff neck, or a stiff knee, wrist, or ankle, in chorea, in tonsillitis, in an evolution of nodules, but also in every pyrexial condition of any kind arising in a child. Whenever there is a suspicion of rheumatic inflammation, it is wise to enforce absolute rest in bed, even if

no cardiac disturbance be perceptible. Complete physical repose and external warmth are of vital importance.

Cases of slight rheumatism are as a rule treated far too lightly. A little timely care, the avoidance of fresh chill, and of excitement of heart or strain of its muscle, may make all the difference in the degree of cardiac mischief.

Dr. Sibson found that a much larger proportion of patients—more than two to one—treated by rest escaped permanent heart mischief as compared with those allowed free action.

In cases of active pericarditis, accompanied by pain, tenderness, and distress, the application of an ice-bag, as recommended by Dr. Lees, is usually grateful, relieves pain, and appears to exercise a favourable influence on the inflammatory process. One or two leeches may be applied to the præcordia, but anything like profuse extraction of blood by leeches is injurious. Children bear loss of blood ill. In cases of persistent, recurrent, intractable endocarditis and pericarditis, which form such sad and especial features of the acute rheumatism of children, digitalis, strophanthus, strychnia, and opium are the drugs of most service. The latter is best given in the form of nepenthe, and in frequent small doses of half a drop to five drops, according to age. More than any other remedy it relieves distress, lessens dyspnœa, and procures sleep. If given with care and discretion no harmful effect need be anticipated. Alcoholic stimulants help by their sedative influence as well as by increasing cardiac power. When the embarrassed heart, by feeble beat, small irregular pulse, and tendency to syncope, shows signs of failure, hypodermic injections of liquor strychniæ in doses of $\frac{1}{8}$ of a minim to 1 minim or more, according to age, combined with 5 to 10 drops of brandy, and given every four to six hours, are the most powerful means of sustaining the failing circulation. Or $\frac{1}{2}$ a minim to 3 minims of tinct. of digitalis may be administered in the same way. In the case of children under seven years of age, however, these remedies should not be administered hypodermically, except in conditions of extreme and urgent danger, and then with the greatest caution and watchfulness.

W. B. CHEADLE

REFERENCES

1. ADAMS, J. C. "Acute Rheumatism in an Infant of Six Months," *Trans. Minnesota Med. Journ.* 1885, p. 109.—2. ASHBY, H. *Brit. Med. Journ.* 1886, i. p. 970.—3. ASHBY, H., and G. A. WRIGHT. *Dis. of Children*, 2nd ed. 1896, pp. 390, 497.—4. BABINGTON. *Guy's Hosp. Rep.* 1841, vi. p. 418.—5. BARENSPRUNG, VON. *Charité Annalen*, 1862, x. Hft. i. p. 117.—6. BARLOW, T. "Rheumatism and its Allies in Childhood," *Brit. Med. Journ.* 1883, ii. p. 509; and WARNER, *Trans. Inter. Med. Cong.* 1881, iv. p. 116.—7. BASCH. "Ein Fall von Polyarthritis Rh. Acuta in der 13ten Lebenswoche." *Prag. med. Wochenschr.* 1884, ix. p. 450.—8. BEGBIE. *Edin. Monthly Journ. Med. Sci.* 1847, vii. p. 740; "Remarks on Erythema Nodosum and its Connection with the Rheumatic Diathesis," *Ibidem*, 1850, 3rd ser. i. p. 497.—9. BOHN, GERHARDT. *Handbuch d. Kinderkrank.* 1877, ii. p. 268.—10. BOKAI, J. *Jahrbuch f.*

Kinderheilkunde, 1885, xxiii. p. 305.—11. BOTREL. *De la Chorée considérée comme affection Rh.* Thèse de Paris, 1850.—12. BOUCHARD, J. B. "Chorée et rh. articulaire aigu: paraplégie consécutive," *Rev. mens. cl. mal. de l'enf.*, Paris, 1888, vi. p. 551; 1889, vii. p. 16.—13. BRIGHT. *Med.-Chir. Trans.* 1839, xxii. p. i.—14. BURY. *Brit. Med. Journ.* 1883, ii. p. 516.—15. CALDWELL. "Acute Rheumatism at Two Months," *Louisville Med. News*, 1883, xvi. p. 364.—16. CAVAFY. *Path. Soc. Trans.* 1883, xxxiv. p. 41; also *Clin. Soc. Trans.* 1883, xvi. p. 43.—17. CHAPIN. "Rheumatism in Early Life," *Boston Med. and Surg. Journ.* 1886, cxiv. p. 231; also *Med. Rec. N. Y.* 1886, i. p. 230.—18. CHEADLE, W. B. "Rheumatic Heart Disease in Children," *Lancet*, 1885, ii. p. 703; "A Clinical Illustration of Certain Phases of the Rheumatic Diathesis," *Lancet*, 1886, i. p. 433; "On the Various Manifestations of the Rheumatic State as exemplified in Childhood and Early Life," *Harveian Lectures* 1888, Lond. 1889; also *Lancet*, 1889, i. pp. 821, 871, and 921; *Cyclopædia* (Keating's) *of the Diseases of Children*, 1889, i. p. 785; *Brit. Med. Journ.* 1896, i. p. 65.—19. COULAND, CAMILLE. *De l'Erythème Papuleux dans ses Rapports avec le Rh.* Paris, 1875. 20.—CRANDALL, F. M. "Rheumatism in Children," *Internat. Clin. Phila.* 1893, 3rd ser. i. p. 176.—21. DASS, A. P. "Acute Articular Rheumatism in a Child Five Months Old," *Med. Reporter*, Calcutta, 1894, iv. p. 74.—22. DAVILLE, ERNEST P. *Chorée et Rhumatisme.* Paris, 1889.—23. DE BEURMANN. "Deux observations d'erythème Rh.," *Arch. gén. de Méd.* 1881, p. 721.—24. DESCROIZELLES. "Rh. aigu infantile," *Journ. des Conn. Med. Prat.* 1885, p. 72.—25. DICKINSON, W. H. *Med.-Chir. Trans.* 1876, lix. p. 1.—26. DONKIN. *Diseases of Childhood*, 1895, pp. 209 and 297.—27. DUCKWORTH, Sir D. *Brit. Med. Journ.* 1883, i. p. 868; *Trans. Clin. Soc.* 1882-83, xvi. p. 190.—28. EADE, Sir P. "A Case of Chorea followed by Erythema and Acute Rh.," *Brit. Med. Journ.* 1889, i. p. 700.—29. FEDELE, N. *Su cinque casi di peritonite reumatica acuta in bambini, con guarigione*, Morgagni, Milano, 1892, xxxiv. part i. p. 125.—30. FUTCHER. "A Study of Subcutaneous Fibroid Nodules," *Johns Hopkins Hospital Bulletin*, Nos. 54, 55, Oct. 10, 1895.—30*a*. GARROD, A. E. "On the Relation of Erythema Multiforme and Nodosum to Rheumatism," *St. Bart. Hosp. Rep.* 1888, xxiv. p. 43; *Med.-Chir. Trans.* 1889, lxxii. p. 145; *Lancet*, 1889, ii. p. 1051; *Treatise on Rheumatism*, 1890.—31. GEE, S. J. "Rheumatic Fever without Arthritis," *St. Bart. Hosp. Rep.* 1888, xxiv. p. 20.—32. GOODHART, J. F. "On the Rheumatic Diathesis in Childhood," *Guy's Hosp. Rep.* 1881, xxv. p. 103; *Diseases of Children*, 1894.—33. GRESWELL, D. A. *A Contribution to the Natural Hist. of Scarlatina*, 1890.—34. HENOCH. "Lectures on Children's Diseases," *Syd. Soc. Trans.* vol. ii.; *Vorlesungen über Kinderkrankheiten*, 2nd ed. 1883, p. 723; *Charité Annalen* (1880), 1882, Jahrgang vii. p. 645. —35. HERRINGHAM, W. P. *Med.-Chir. Trans.* 1889, lxxii. p. 117.—36. HILLIER. *Med. Times and Gaz.* 1863, ii. p. 142; *Diseases of Children*, 1868, p. 238.—37. HUGHES. *Guy's Hosp. Reports*, 1846, 2nd series, iv. p. 360.—38. IMMERMANN. Ziemssen's *Handbuch*, 1876, xiii. ii. p. 447.—39. JACOBI. "Acute Rheumatism in Infancy and Childhood," Seguin's *Amer. Clin. Lectures*, i. No. 11.—40. KINNICUTT. *American Arch. of Dermatol.* 1875, i. p. 192.—41. LEGG, W. *St. Bart. Hosp. Rep.* 1883, xix. p. 177.—42. LEWIS, M. J. "A Study of the Seasonal Relations of Chorea and Rheumatism for a Period of Fifteen Years, 1876 to 1890 inclusive," *Trans. Ass. Amer. Physicians*, Phila. 1892, vii. p. 249; also *Amer. Journ. Med. Sci.* Phila. 1892, New Ser. civ. p. 251.—43. MACKENZIE, S. *Trans. Internat. Med. Cong.* Lond. 1881, iv. p. 97; *Trans. Clin. Soc.* 1882-3, xvi. p. 188; *Collect. Invest. Record*, vol. iii.; "An Address on some Points regarding Acute Rh. requiring Investigation," *Brit. Med. Journ.* 1886, i. p. 99; "On Erythema Nodosum, etc.," *Clin. Soc. Trans.* 1886, xix. p. 215.—44. MACKAY, E. "Chorea; Rheumatism; many large Subcutaneous Nodules; Mitral Reflux," *Lancet*, 1894, i. p. 145.—45. MEYNET. *Lyon Méd.* 1875, p. 495.—46. MONEY, ANGEL. *Brain*, 1882-83, vi. p. 511; "Rheumatism in Children," *Lancet*, 1886, ii. p. 158; "Nodular Periostitis in Children, Rheumatism, and Heart Disease," *Ibid.* 1889, ii. p. 265.—47. OSLER. *Med. News* (Phila.), 1887, ii. pp. 427-465; also *Amer. Journ. Med. Sci.* 1887, xciv. New Ser. p. 374; also on "Visceral Complications of Erythema, Exudativum multiforme," *Amer. Journ. Med. Sci.* Dec. 1895.—48. PETER. *France méd.* 1886, ii. p. 1037.—49. REHN, J. H. *Verhandlungen des Cong. f. i. Med.* 1885, iv. p. 296; Gerhardt's *Handbuch der Kinderkrankheiten*, 1878, Bd. iii. Hft. i. p. 22 and p. 268.—50. ROGER. *Gaz. des Hôp.* 1870, xliii. p. 257; *Arch. gén. de Méd.* 1886, 6th series, viii. p. 641.—51. SANSOM, A. F. "Clinical Lectures on Diseases of the Heart in Children," *Med. Times and Gaz.* 1879, ii. pp. 227-255, etc.—52. SCHÖNLEIN. *Allg. u. spec. Pathol. u. Therap.* 1837, ii. p. 45.—53. SCHWARTZ. *Wiener med. Wochenschr.* 1883, xxxiii. p. 991.—54. SCUDAMORE, Sir C. *On Rheumatism*, 1827.—55. SÉE, G. "Chorée

et rhumatisme," *Méd. Mod. Par.* 1891, ii. p. 733; *Ibid.* 1892, iii. pp. 25, 42, and 73.—56. SENATOR. Ziemssen's *Handbuch der spec. Pathol. und Therap.* 2nd edit. 1879, xiii. Hft. i. p. 1.—57. SIMON, J. "Du rhumatisme chez les enfants et son traitement," *Bull. Méd. Par.* 1893, vii. p. 1093; also *Écho Méd. Toulouse*, 1894, 2nd ser. viii. pp. 49-61.—58. STURGES, O. *Trans. Internat. Med. Cong.* Lond. 1881, iv. p. 105; *On Chorea*, 2nd ed. 1893; Lumleian Lectures, "Heart Inflammation in Children," *Brit. Med. Journ.* 1894, i. pp. 505, 561, and 623.—59. SYERS, H. W. "Chorea and Rheumatism," *Lancet*, 1889, ii. p. 1271.—60. TODD. *Clin. Lectures*, 1854, p. 428.—61. TOWNSEND, F. M., CRANDALL, ADAMS, S. S., and others. *Trans. Amer. Pædiat. Soc.* (1892), 1893, iv. p. 215.—62. TROUSSEAU. *Clinique méd.* 1860, i. p. 17.—63. WHIPHAM, T. T. "Collect. Invest. Rep." *Brit. Med. Journ.* 1888, p. 387.

W. B. C.

CHRONIC RHEUMATISM

THE term chronic rheumatism, as usually employed, is a survival from the days when scarcely any attempt had been made to distinguish the various diseases which tend to implicate the joints; and when we try so to limit its use that it may acquire some degree of scientific accuracy, we find that the great majority of the morbid conditions thus described are not in a strict sense rheumatic at all; and further that, as we shall see, those very cases in which the evidence of the continuous smouldering activity of the true rheumatic process over a period of months or even years is clearest are not usually included as examples of chronic rheumatism.

In the first place, it is necessary to eliminate from the category under discussion all examples of chronic articular gout, and of rheumatoid arthritis; as well as those in which chronic joint lesions are associated with gonorrhœa, syphilis, or hæmophilia. There remains a group of cases in which chronic articular changes have their origin in true rheumatism, but in many of these the lesions are rather of the nature of sequels than of active manifestations of the rheumatic process. It is true that there are those who demur to the exclusion of rheumatoid arthritis from the group; but the general tendency at the present time is to regard rheumatoid arthritis and true rheumatism as maladies essentially distinct and widely differing in their pathology.

By true rheumatism is here meant that systemic disorder which, although it manifests itself most typically in an attack of rheumatic fever, does not necessarily give rise to arthritis, but limits its effects in some instances, and especially in children, to the production of such phenomena as chorea, erythema, peri- and endocarditis or fibrous nodules. That this disease sometimes assumes a chronic form is beyond question; and, like syphilis and ague, its continued activity may manifest itself, now in one form, now in another, during a very long period.

Bearing this fact in mind, we may group the cases to which the name of chronic rheumatism is more or less correctly applied under three heads, as follows:—

1. *Frequently recurring slight articular attacks, in rheumatic subjects.*

2. *Cases of chronic abarticular rheumatism, a condition best seen in children.*

3. *Joint lesions which persist after an attack, or succession of attacks, of acute or subacute rheumatism, and in some instances lead to permanent damage of the structures involved.*

It will be well to consider these three classes of cases separately.

1. Cases of the *first kind* are extremely common; whereas some patients who suffer from a single attack of acute rheumatism are free for the

remainder of their lives from any further manifestations of the disease, others have repeated acute or subacute attacks separated by intervals of complete immunity; others again are seldom free from articular pains for any considerable length of time. A similar condition is not uncommon in those who come of rheumatic families, but who have never themselves suffered from any of the more acute manifestations of the disease.

In such cases the body temperature is seldom appreciably raised during the attacks, nor is there any noticeable swelling or redness of the joints which are the seats of pain. Pain and stiffness, sometimes accompanied by creaking on movement, are as a rule the only symptoms; and the tendency of the affection to shift from joint to joint, so conspicuous in acute cases, is often noticed. After lasting for a few days the pain may pass away as quickly as it came; but recovery is sometimes delayed because the trouble is not sufficiently severe to enforce rest of the parts.

It is seldom that adults present cardiac lesions in connection with such attacks; and but for the discomfort which they cause they are of little importance, save that they afford a warning of a special liability, and, unless proper precautions are taken, may culminate in rheumatic fever. As a rule such articular pains yield rapidly to salicylic treatment.

It is by no means easy to ascertain how far the "rheumatics" so often complained of by elderly patients, and especially by women of the working-classes who follow laborious occupations and are much exposed to cold and damp, are of this nature. It seems probable that, as Dr. Maclagan believes, in some of these cases there results from exposure a simple inflammatory condition of joints which had been damaged by antecedent rheumatism, and had thereby become points of diminished resistance; and, further, that the efficacy or failure of salicylic treatment affords a rough test of the class into which the case properly falls. Certainly in a considerable number of such cases a history of acute rheumatism at some earlier period is forthcoming; and Senator aptly compares them with cases in which chronic bronchitis has its origin in the acute bronchitis of measles.

Such rheumatic pains are profoundly influenced by weather and seasons, and are apt to recur annually with the return of autumn; but sometimes, on the other hand, the summer is the period of greatest suffering.

I once tabulated a series of forty examples of this condition among hospital out-patients. Of the forty patients nine gave a clear history of rheumatic fever in their near relations, three others of rheumatism and heart disease, and six of "rheumatism." Sixteen patients had themselves suffered from acute rheumatism, and two others from chorea. In eleven cases only was there neither family nor personal history of the more acute forms of the disease. The only point to lay stress upon is this, that no less than two-fifths of the patients had themselves suffered from rheumatic fever; for the more one attempts to elicit family histories from uneducated and even from educated people, the more sceptical does one become of the value of the information obtained.

In the "rheumatics" of elderly people the larger joints are chiefly

affected, and, as in so many other forms of joint disease, the knees being the joints subject to the greatest stress, show a special liability.

Creaking and crackling in the affected joints is very common, and can usually be distinguished from the grating of the exposed bony surfaces obtained in advanced cases of rheumatoid arthritis. The crackling may be well heard with the stethoscope, but the auscultation of joints which I have practised in a large number of cases has not appeared to afford any information of diagnostic value.

In only five of my forty patients was there distinct evidence of valvular heart disease, and four of these gave histories of antecedent attacks of acute rheumatism from which the lesions in all probability dated. When it is considered that even in acute rheumatism the liability to endocarditis rapidly diminishes with advancing age, it is to be expected that in such cases the liability to fresh cardiac lesions will be practically *nil;* this has certainly been my experience, which is, however, at variance with the statements of Besnier and some other French physicians, who hold that, even in chronic cases, a slowly progressive subacute endocarditis is by no means uncommon.

2. The cases of the *second kind* are those to which the name of Chronic Rheumatism is most strictly applicable. As already mentioned, this form of the disease is most frequently met with in children, but it is not very uncommon in early adult life. Without any considerable febrile disturbance the rheumatic process continues to manifest itself now in one form, now in another, never leaving the patient free.

Any one who has had experience of rheumatism in children will at once recall cases of this description, in which a child has exhibited one rheumatic lesion after another, now chorea, then an erythematous eruption, then fresh cardiac mischief or articular pains; meanwhile successive crops of subcutaneous fibrous nodules are constantly appearing, fresh ones springing up before those which preceded them have disappeared. Meanwhile the child becomes more and more anæmic, a veritable rheumatic cachexia is established, and he ultimately succumbs to a more acute exacerbation, which often takes the form of pericarditis.

The following case is a good example of this condition :—

The patient, a boy, first came under observation at the age of seven years, suffering from articular pains, followed by chorea; and during his stay in hospital numerous subcutaneous nodules and an eruption of erythema marginatum appeared. Six months later he was readmitted with similar symptoms, the nodules were still present, and the chorea and erythema returned. He has now reached the age of eleven years, and has a well-marked presystolic thrill and a loud presystolic murmur at the cardiac apex; but (and this is the remarkable feature of the case) from the time of the first attack, now four years ago, the boy has never been free from subcutaneous nodules, fresh crops of which have constantly appeared, replacing others which have disappeared. At one time, when he was eight years old, he exhibited more nodules than any other patient I have ever seen, not only in the usual situations, but also

all over the back and abdominal walls. At one time he was very anæmic, but of late his condition has decidedly improved in this respect.

Surely these are the cases most strictly entitled to the name of chronic rheumatism.

3. The articular lesions which are met with as *sequels of acute rheumatism* are of various kinds. In some cases pain and stiffness of the affected joints persist for a considerable time, but ultimately yield completely to time and appropriate treatment.

Occasionally a single joint among the many which have been affected during an acute attack, instead of recovering like the rest, becomes the seat of a chronic synovitis, and may ultimately become completely fixed by fibrous ankylosis. More commonly a minor degree of stiffness remains, which yields to passive movements under an anæsthetic. In an interesting paper C. von Noorden has shown, and experience confirms his statement, that such accidents are especially apt to occur, when rheumatic fever appears as a complication of pregnancy or of the puerperal state.

Among the occasional sequels of acute rheumatism a place must be assigned to rheumatoid arthritis (see p. 75); but other conditions are met with under like circumstances which more or less closely simulate that disease. When the hands have been affected during the acute attack, a very extreme degree of ulnar deflexion of the fingers may result—a condition never produced by muscular contraction alone, but implies past or present affection of the metacarpo-phalangeal joints. In the rheumatic cases which present this deformity, the recovery of the joints themselves is apparently complete, but the extensor tendons may slip from their grooves when the fingers are bent.

The finger-joints may present fusiform enlargements, which may disappear under observation, or may be permanent. Such enlargement may result from thickening of the joint capsule; and although the resemblance to rheumatoid arthritis is so striking, the cartilages and bones may show no morbid change post-mortem.

Jaccoud first called attention to this condition, and assigned to it the name of *Rhumatisme Fibreux*. The first accurate description of the condition of the joints post-mortem was published by two Italian observers, Drs. Brigidi and Banti. It is probable that some of the very puzzling chronic joint affections met with in children are of this nature—such, for example, as have been described by Drs. Barlow, Pasteur, and others; but our knowledge of such chronic arthritic changes of childhood is as yet in its infancy. [*Vide* p. 106.]

The immunity of the temporo-maxillary joints, and the absence of any distinct lipping or osteophytic out-growths, are points which may aid in the diagnosis of this "fibrous rheumatism" from true rheumatoid arthritis; but even when every care is taken a certain diagnosis may sometimes be impossible, at any rate for a time; and the resemblance between the two maladies is increased by the fact that, in both, the atrophy and contracture of the muscles which control the affected joints produce deformities of similar kinds.

Treatment of chronic rheumatism.—The slight articular attacks which frequently recur are best treated by means of salicylates; but in more chronic cases these drugs have often no influence upon the joint lesions; and even during the convalescent period of rheumatic fever a stage is not infrequently reached in which the salicylates appear to be of no more service. Under these circumstances I have found the alkaline quinine treatment the most effectual: the sulphate of quinine suspended by means of mucilage is given in doses of 3-5 grains with an alkali; iodide of potassium may usually be added with advantage. In some cases guaiacum is of service, and local applications of iodine or of some liniment are often useful. When one or more joints pass into a state of chronic synovitis, treatment by blisters as well as by splints, and other surgical means, may be required; care being taken that if the joint should become fixed the position of the limbs should be the most advantageous possible. Passive movements under an æsthetic, and in slighter cases mechanical exercises, will be of service in removing the minor degrees of stiffness.

External treatment by mineral baths will often prove of the greatest service in removing the residual pain and stiffness after an acute attack, and also the still more chronic articular pains. Brine baths, such as those of Droitwich, the douche-massage treatment initiated at Aix-les-Bains, and local steam baths, are especially serviceable; but cases of this kind are among those which derive benefit from treatment at almost any spa where external treatment is carried out, whether in the ways above mentioned or by simple hot immersion baths.

When the articular pains return regularly with the onset of cold weather, the patient should be advised to winter in a climate warmer than our own. Patients who suffer in this way should be properly clothed in woollen garments, and they often derive great relief from a woollen covering specially adapted to the affected joint.

ARCHIBALD E. GARROD.

REFERENCES

1. BRIGIDI and BANTI. *Lo Sperimentale*, 1880, xlv. p. 604.—2. BESNIER, E. *Dict. Encyclop. des Sciences méd.*, article "Rhumatisme," 1876.—3. JACCOUD. *Leçons de clin. méd.* 1867, p. 598.—4. MACLAGAN. *Rheumatism*, 1881, 2nd ed. 1896.—5. NOORDEN, C. VON. *Charité Annalen*, 1892, xvii. p. 185.

A. E. G.

MUSCULAR RHEUMATISM

PAINS referred to the muscles are met with in the course of a variety of diseases, and, apart from any considerable degree of constitutional disturbance, frequently arise as the result of exposure to cold and damp. It is to myalgia of this latter kind that the application of the name of muscular rheumatism is restricted. In this connection the word rheumatism is used in its looser sense, as indicative merely of an origin in exposure; for although sufferers from true rheumatism in its acute and subacute forms frequently complain of muscular pains in addition to the articular, there is little evidence that muscular rheumatism stands in any direct relationship to that disorder or has a common origin.

With gout, on the other hand, the so-called muscular rheumatism frequently has an obvious relationship, and this is especially the case with that variety in which the lumbar muscles are involved. The alternation or association of lumbago with sciatica and articular gout is a matter of common experience. On the other hand, there is a large remnant of cases in which the gouty element can apparently be excluded, and in which the only obvious cause at work is direct exposure to cold or damp. It is probable that, in the course of time, it will become possible to adopt some more accurate system of classification even in these cases, and that the symptom myalgia will cease to occupy a distinct place in the catalogue of diseases.

Our knowledge of muscular rheumatism is entirely based upon clinical observation, for the affection has fortunately no fatal tendency; thus we are compelled to glean our knowledge of the processes at work in the muscles and associated fibrous structures from external examination alone. From such external examination little can be learned; for when there is evidence of active inflammatory changes in the muscles the condition is described as myositis, and no longer as muscular rheumatism; the latter name being reserved for those cases in which such objective signs are wanting.

Among the various pathological explanations which have been suggested those have most claim to attention which ascribe the muscular pains to active hyperæmia, to a neuralgic affection of the terminations of the nerves in the muscles, or to slight inflammatory changes in the fibrous elements which enter into the structure of the muscles. In favour of the inflammatory theory is the fact that in some long-standing cases actual swellings arise in the course of the affected muscles, the "Rheumatische Schwiele" of German authors.

Pain is the most prominent symptom of muscular rheumatism, and this pain is characterised by its very great increase when the affected structures are brought into use or placed upon the stretch. This character is of great diagnostic value, as it serves to distinguish true myalgia from

a variety of other painful conditions which not uncommonly attack the same parts of the body, and with which it is apt to be confused. In slight cases there may be little or no pain while the patient is completely at rest; but in acute cases he is never at ease whatsoever position he may adopt. Tenderness of the affected muscles is another important symptom often of use in diagnosis, especially in the more acute cases.

Muscles which have become the seats of muscular rheumatism are frequently in a condition of spasmodic contracture, a feature which is especially well marked in cases of stiff-neck; in it the sterno-mastoid is the affected part, and the condition is known as rheumatic torticollis.

The mode of onset varies greatly in different cases. In the most acute examples the onset is extremely sudden, the patient being, as he says, "suddenly set fast with lumbago"; but in chronic cases it is gradual and insidious. The duration of the attack is also very variable. In acute cases the pain, although very severe, may last but a few days, or a week; whereas in chronic cases it may persist for months or even for years, the patient being never free from myalgia in some part of his body or limbs.

Occasionally slight swelling is observed in the region of the affected muscle, but this is not usually the case.

The constitutional symptoms associated with muscular rheumatism are as a rule slight, and are often entirely wanting. The temperature may be normal or slightly raised, and the urine may deposit pink uratic sediment; but, as a rule, the patient feels little illness beyond the intense pain which he suffers especially on movement, though it is often increased by the warmth of bed.

Usually a smaller or larger group of muscles functionally related to each other is alone involved, and certain such groups are specially liable to suffer. Hence, for want of a better system, the classification of muscular rheumatism generally adopted is a topographical one. Thus we speak of lumbago when the lumbar muscles are the seat of pain, of intercostal rheumatism, of rheumatic torticollis, and so on. The different age incidence of the several varieties affords further grounds for such a classification, and probably indicates differences of pathology.

In many cases the association with exposure is direct and evident—exposure to a draught or sitting in damp clothes often preceding by a few hours or a day or two the onset of the symptoms; and the pain, at least in the earlier stages, may even be confined to that portion of the body which has been in direct contact with the wet clothing. Such an exciting cause is by no means incompatible with a gouty or other constitutional origin; and just as injury to a joint may determine an attack of articular gout, so an exposure may give rise, in a gouty subject, to an attack of lumbago or of sciatica. Another potent exciting cause is an injury, which is usually of the nature of overstrain, either sudden or prolonged; and, like cold and damp, such injury may serve as the immediate cause when the remoter causes lie in a peculiar constitutional state.

It will be necessary to refer briefly to the special characters of the more important varieties of muscular rheumatism, and of these lumbago may well take the leading place.

Lumbago.—All that has been said of the ætiology and symptoms of muscular rheumatism in general applies to this affection. It is commoner in men than in women, and is almost confined to adult life—characters which are due, at least in part, to its frequent association with gout. It is distinguished from other forms of lumbar pain, such as result from constipation, small-pox, etc., by being greatly increased by movement, and especially by stooping; from the pain of renal gravel by the same feature, and by the absence of indications of renal affection in the urine; and from the pains due to diseases of the spinal column and its meninges by the absence of objective signs of spinal disease, and of the radiating pains of meningitis.

Intercostal rheumatism has to be distinguished from neuralgia of the intercostal nerves, whether primary or secondary to spinal diseases; from the pain of herpes zoster, of pleurisy, and of diseases of the ribs. These distinctions do not as a rule offer much difficulty. The most frequent cause is a traumatic one, namely, the strain produced by severe and frequent coughing; hence the affection is commonly met with in sufferers from chronic bronchitis.

Rheumatic torticollis is especially an affection of childhood, and is usually directly attributable to a chill. It has to be distinguished from pains due to rupture of fibres of the sterno-mastoid, as in the hæmatoma of infants; from stiff-neck due to diseases of the cervical spine, such as caries and gonorrhœal or rheumatoid spondylitis; and from meningeal affections such as cervical pachymeningitis. In cases of rheumatic torticollis there is conspicuous muscular spasm; one or both sterno-mastoids may be affected.

Other varieties of muscular rheumatism are sometimes distinguished by special names; such as omalgia when the shoulder muscles are affected, cephalalgia, and the like.

Treatment of muscular rheumatism.—It is in acute cases that the most satisfactory results are obtained from treatment; the more chronic cases are apt to prove very refractory.

Among internal remedies sodium salicylate is sometimes of great service, the pain, especially in some cases of acute lumbago, yielding to this drug almost as rapidly as the articular pains of acute rheumatism itself. In other cases most benefit is derived from quinine in considerable doses, administered in suspension with an alkaline salt and iodide of potassium. In chronic cases guaiacum has appeared to me to be the most useful drug, and its efficacy is apparently increased by the simultaneous administration of an alkali. In the acute stages a hot bath or a vapour bath sometimes gives marked relief. Of local applications, belladonna liniment on spongio-piline is as efficacious as any. When the pain is very intense it may be necessary to administer morphia in the early stages, either by the mouth or by local subcutaneous injection.

The patient should adopt the position of greatest ease, his food should be light, and the bowels should be kept freely opened.

In chronic cases the pain is seldom so severe as to call for urgent measures of relief. Great benefit is frequently derived from mineral water treatment; and brine baths, such as may be taken at Droitwich, Nantwich, and a few other places in this country, and at various places on the Continent, are especially serviceable. In gouty cases especially, the various watering-places, such as Bath, Buxton and Harrogate which in our own country enjoy a reputation for the successful treatment of gouty conditions, may be visited with benefit; and the regimen and other therapeutic measures usually prescribed for the gouty should be insisted upon. [See arts. on "Balneo-therapeutics," "Dietetics," and "Electricity."]

Electricity and other special forms of local treatment are useful in some cases; and it stands to reason that patients liable to lumbago and other forms of muscular rheumatism should wear woollen underclothing both in summer and winter, and should be specially careful to avoid any such exposure or chill as may precipitate an attack.

ARCHIBALD E. GARROD.

REFERENCES

1. BEAU. *Archives gén. de médecine*, 1862, 5e sér. xx. p. 641.—2. BOUCHARD. *Dict. encyclop. des sci. méd.* 1870, article "Lumbago."—3. DESNOS. *Dict. méd. et chir. prat.* 1875, xx. p. 744.—4. FRORIEP. *Die rheumatische Schwiele*, 1843.—5. VALLEIX. *Bull. gén. de thérap.* 1848, xxxv. pp. 296, 385.—6. VIRCHOW. *Archiv f. pathol. Anatomie*, 1852, iv. p. 266.

A. E. G.

GONORRHŒAL RHEUMATISM

THE association of inflammation of joints with gonorrhœa, which has long been a recognised clinical fact, was first described by Selle and Swediaur in the year 1781. Since then this form of arthritis has been carefully studied by many observers, who have expressed widely different opinions as to its nature and pathological relationships. There is no longer any doubt that the so-called gonorrhœal rheumatism is essentially distinct from rheumatism proper; and some modern authors, discarding the time-honoured name as misleading, prefer to speak of gonorrhœal arthritis or synovitis: yet these terms are themselves not free from objection, seeing that they express but a part of the truth; for, just as rheumatism and gout are systemic diseases of which arthritis is merely the most conspicuous of many manifestations, so also the arthritis of gonorrhœa is but one of several forms in which a systemic infection gives evidence of its activity.

Under the name of gonorrhœal rheumatism I propose to describe a

systemic malady which follows local gonorrhœal infection; which shows itself most frequently by the production of acute inflammatory lesions of one or more joints, less frequently by the production of a non-purulent conjunctivitis, or of iritis, and of painful affections of muscles and fasciæ.

It would be more scientific to describe this group of phenomena among the complications of gonorrhœa rather than as a distinct disorder; but, from the point of view of convenience, the alternative plan has much to be said for it, as the physician often has occasion to diagnose gonorrhœal rheumatism from the other diseases in which joint lesions play a conspicuous part.

Ætiology.—The necessary precursor of an attack of gonorrhœal rheumatism is a gonorrhœa; but it is not necessary that the primary infection should be urethral, and articular lesions obviously of this nature have occasionally followed inoculation of the conjunctiva. Thus Poncet of Cluny has recorded a remarkable case in which the separate inoculations of the conjunctivæ of the two eyes for the cure of granular lids was on each occasion followed by an attack of gonorrhœal rheumatism; and Lapersonne on the authority of Testelin cites similar cases. Again Clement Lucas, Deutschmann, Lindemann, Höck and others have described joint lesions of this nature in infants suffering from purulent conjunctivitis.

The interval which elapses between the gonorrhœal infection and the appearance of the arthritis varies greatly in different cases. In some instances the interval is as short as a week; but three or four weeks usually elapse, and there appears to be no doubt that the period is occasionally extended to several months, when a slight gleet may be the only remaining evidence of the local trouble. In Poncet's case the arthritis followed a month after the inoculation of the first eye and seven days after that of the second.

The influence of external conditions in determining the occurrence and seat of the local manifestations of systemic diseases is apparently very potent; and it does not seem improbable, as some authors have maintained, that cold and damp have an influence in provoking the outbreak of gonorrhœal arthritis; but in most cases no satisfactory evidence of the action of such concurrent causes is forthcoming.

Sex.—It seems certain that men are far more liable than women to the affection under discussion; but it is certainly not the case that the latter sex enjoys immunity from it. Cases are not very uncommon in which a joint affection, having all the characters of gonorrhœal rheumatism, accompanies a purulent vaginitis; and it seems probable that the number of cases in which this diagnosis is arrived at is diminished by the difficulty of ascertaining the fact of the original affection.

The *age* of greatest liability is, of course, that at which gonorrhœa itself is chiefly acquired; and the cases in which gonorrhœal arthritis has followed ophthalmia neonatorum show that the liability is present even in earliest infancy.

The disease has no tendency to *relapse* after the gonorrhœa has been effectually cured, unless there be a fresh infection—under which circumstances a patient who has once suffered from gonorrhœal rheumatism is almost certain to have another attack; on the other hand, so long as any gleet remains the patient is not free from the risk of a relapse.

Symptoms.—The general constitutional symptoms of gonorrhœal rheumatism are slight. The temperature is usually little if at all raised, and the other symptoms which attend febrile disturbance are consequently wanting. Febrile disturbance of a hectic type is sometimes met with, such as occasionally complicates gonorrhœa apart from any of the local phenomena under discussion.

The articular lesions.—Gonorrhœal rheumatism shows a tendency to attack the larger rather than the small joints; it may be limited to a single articulation, or may involve almost every joint in the body. Certain joints which are little liable to suffer in other forms of articular disease not infrequently suffer, and among these the temporo-maxillary articulations occupy a prominent place. These joints enjoy an almost complete immunity from gout and rheumatism, but are, on the other hand, among the seats of election of rheumatoid arthritis. The sacro-iliac and sterno-clavicular articulations are also liable to invasion.

The following table, compiled from those of Foucart, Brandes, Rollet, and Fournier, gives the frequency of the affection of the several joints in a total of 119 cases:—

Knee	attacked in	83	cases.	Temporo-maxillary joint	attacked in	6 cases.
Ankle	,,	32	,,	Metatarsus and Tarsus	,,	5 ,,
Fingers and Toes	,,	23	,,	Sacro-iliac joint	,,	4 ,,
Hip	,,	16	,,	Sterno-clavicular	,,	3 ,,
Wrist	,,	14	,,	Chondro-costal joints	,,	2 ,,
Shoulder	,,	12	,,	Tibio-fibular joint	,,	1 case.
Elbow	,,	11	,,			

The special liability of the knee is very conspicuous; but this joint is especially liable to arthritis of all kinds, and even in rheumatoid arthritis is only less frequently attacked than the small joints of the fingers. On this point the statistics merely confirm our every-day experience.

The outset of the arthritis is marked by swelling and pain. The swelling is due in large measure to the effusion of fluid into the cavity of the joint, or into the adjacent bursæ and tendon sheaths; as a rule there is no conspicuous blush over the affected part, and but little local increase of temperature. These phenomena may be present, however, in extreme cases. The pain, though often severe, is apparently less than that of either gout or rheumatism; at any rate, the patient allows more free handling and movement of the parts without complaint. Some who have suffered from both true rheumatism and gonorrhœal arthritis describe the pains in these two diseases as of different characters. In exceptional cases, on the other hand, the pain is extremely severe, and the effusion into the cavity of the joint may be so excessive as to call for paracentesis.

The inflammation tends to persist in the joints first attacked, and

there is an entire absence of the tendency to shift from joint to joint, which is so conspicuous a feature of the arthritis of acute rheumatism.

Suppuration is a rare, although by no means an unknown event. As a rule the affected joints recover completely although slowly; but there is a conspicuous tendency in this disease to the formation of fibrous adhesions, and in order to restore the mobility of the articulation it may be necessary to have recourse to passive movements under an anæsthetic.

In some very bad cases the patient is left in a pitiable condition, with many joints in a state of almost complete fibrous ankylosis; but this seldom occurs except as the result of repeated attacks, each in turn provoked by a fresh gonorrhœal infection. Another occasional event is the supervention in the affected joints of the changes of rheumatoid arthritis, which may follow upon this as upon other forms of arthritis, such as that of rheumatism and gout. Such cases, in which there is obvious bony enlargement and osteophytic formation, must be carefully distinguished from those in which there is simply fibrous ankylosis.

Effusion into the tendon sheaths is common in association with arthritis of the wrist or ankle; when this is present the dome-like swelling of the dorsum of the hand may readily lead to a mistaken diagnosis, as it bears a striking resemblance to the condition so often seen in gouty cases.

Affections of fasciæ play a conspicuous part in gonorrhœal rheumatism. In the feet there is often pain referable to the plantar fascia, especially in the neighbourhood of the os calcis; and it is a well-known clinical fact that this disease and gout are the most frequent causes of persistent pain in the neighbourhood of the heel. The ligaments of the foot are apt to yield, and, when the joints of the foot have been involved, flat foot is a common sequel of gonorrhœal as it is of true rheumatism. The lumbar fascia is another common seat of pain, as also are the other fasciæ of the trunk; and this fact may often afford valuable aid in arriving at a correct diagnosis in doubtful cases.

Affections of muscles.—Myalgia is common in cases of gonorrhœal rheumatism, and occasionally the muscles are so much more gravely involved as to exhibit the signs of an acute myositis. The following case afforded an excellent example of this rare condition:—

J. E., aged 20, had acquired gonorrhœa three months before he was first seen. Five weeks from its onset he had pain in both knees and in the left shoulder-joint. When he presented himself in the Casualty Department of St. Bartholomew's Hospital there were signs of effusion in the left shoulder-joint, the left arm was held in the flexed position, and the biceps humeri was swollen and as hard as wood throughout its entire length. The muscle was exquisitely tender, and there was considerable local heat; its inflamed condition precluded any movement at the elbow-joint, which was bent at a right angle, but was not itself involved.

The patient was treated as an out-patient, and the myositis subsided. Three weeks after his first attendance he was admitted under the care of

the late Mr. Morrant Baker, to whom I am indebted for the further notes of the case. At that time he presented a similar affection of the triceps of the other arm, which, unlike the previous myositis, could not be attributed to local extension, as the joints of the right arm were quite unaffected. This also subsided under treatment, but both the muscles involved remained somewhat atrophied. Dr. Sydney Phillips has referred to a similar case which came under his notice, in which there was a like induration and rigidity of the arm muscles in association with gonorrhœa.

Affections of nervous structures.—A painful affection of the sciatic nerve must be mentioned among the occasional phenomena of gonorrhœal rheumatism, but in my experience it has not occurred with any approach to the frequency assigned to it by some authors. Fournier, for example, met with sciatica in no less than five of a series of thirty-nine cases.

In some instances in which the vertebral joints are involved, symptoms due to implication of the nerve-roots are observed; and there are a few cases on record in which spinal symptoms, amounting in some instances to complete paraplegia, have appeared without any recognisable local cause.

Some of these cases have ended in recovery, and in others, which resulted fatally, no post-mortem examination was made. Leyden has recorded a case in which meningo-myelitis was found at the autopsy; and Dr. Gowers describes one in which acute ascending myelitis followed close upon an attack of gonorrhœa.

Ocular lesions.—The affections of the eye in gonorrhœal rheumatism are comparatively common, and take the form of conjunctivitis or sclerotitis, and iritis. The conjunctivitis, which occurs with greater frequency than iritis, must be carefully distinguished from the purulent ophthalmia due to inoculation of the conjunctiva with the gonorrhœal virus. It is present at some stage in a considerable proportion of the cases, and is apparently to be classed on a par with the joint lesions, as one of the manifestations of the systemic infection.

Iritis, when it occurs, usually attacks the two eyes in succession or simultaneously. It is of the serous or "rheumatic" variety.

The following case offers a well-marked example of gonorrhœal rheumatism with ocular lesions:—

H. B., a man of twenty-three years of age, acquired gonorrhœa in June 1887. Three weeks later he had pain in the feet, followed by swelling of the left foot and ankle. Later both knees became swollen. The discharge ceased in October, according to the patient's statement. In January 1888, at which time he first came under observation, the joints were still painful, and there was some degree of flat foot. He then had well-marked conjunctivitis, as well as iritis of the right eye. In February, after the right eye had entirely recovered, left iritis set in.

Fournier met with ocular lesions in fifteen out of thirty-nine cases, but the joints were not usually affected contemporaneously with the eyes. He quotes a case in which four attacks of gonorrhœa within five years were followed, in the first instance, by ocular lesions alone; in the second and

third instances by affections of the eyes and joints, and in the fourth by arthritis alone.

Cardiac lesions.—One of the most striking differences between gonorrhœal and true rheumatism is the practical immunity of the endocardium in the former disease. It is true that cases are on record in the course of which both pericarditis and endocarditis have occurred, but the very rarity of such records serves to emphasise the rule. I have met with a single case in which a murmur, having all the characters of that of mitral regurgitation, appeared in the course of a definite attack of gonorrhœal rheumatism, and disappeared entirely during convalescence. In considering such cases we must be on our guard against including, as organic, hæmic or other murmurs not due to endocarditis; and we must remember also that genuine acute rheumatism may occur in the course of gonorrhœa. These points have been insisted on by Dr. R. L. MacDonnell, of Montreal, in a paper in which he records a case of great interest. Gluzinski, who has collected thirty-one records of cardiac lesions associated with gonorrhœal rheumatism, suggests that the endocarditis, when it occurs, is rather of the ulcerative than of the simple type; and a considerable number of cases are on record in which death has resulted from endocarditis undoubtedly of the infective variety. As these cases have very important bearings upon the pathology of gonorrhœal rheumatism it will be necessary to refer to them later in this connection.

Affections of serous membranes.—The occasional occurrence of pericarditis in the course of gonorrhœal rheumatism has been discussed already, and pleurisy, with or without effusion, must be included among the occasional manifestations of this disease. A particularly convincing example of gonorrhœal pleurisy has been recorded by Bordoni-Uffreduzzi.

Diagnosis.—The presence of active gonorrhœa or of gleet usually renders the diagnosis of gonorrhœal rheumatism an easy matter; indeed the clinical features of the joint lesions themselves often suggest the correct diagnosis, which may be confirmed by the presence of a urethral discharge. On the other hand, it is not safe to conclude that any arthritis in a patient suffering from gonorrhœa is necessarily gonorrhœal.

It is probable that the disease, at any rate in the earlier stages, is not infrequently mistaken for true rheumatism, especially when it occurs in women; but suspicion is soon aroused by the absence of marked fever, or by the obstinacy of the arthritis which shows no disposition to yield to the salicylic treatment.

The occurrence of conjunctivitis or of iritis in the course of a supposed attack of rheumatic fever, or the establishment of arthritis in the temporo-maxillary or sterno-clavicular joints, will always suggest the possibility of a gonorrhœal origin; but it must not be forgotten that rheumatoid arthritis in its more acute forms is also liable to be mistaken for rheumatic fever, and that in this disease the temporo-maxillary joints are even more liable to be involved.

Again, it must be remembered that a purulent urethritis of a non-specific nature is an occasional concomitant of gout; and such urethritis may be mistaken for gonorrhœa, whilst the attendant gouty arthritis may be ascribed to the urethral affection. The characters of the articular lesions which lend aid in the diagnosis of gonorrhœal rheumatism have been already discussed and need not be recapitulated.

Prognosis.—The prognosis on the whole is favourable: a fatal termination is extremely rare; and recovery, although long delayed, is as a rule complete. Nevertheless in giving a prognosis the risk of fibrous ankylosis of the affected joints should always be remembered, and also the possibility of the subsequent development of rheumatoid changes. The patient should be emphatically warned that a fresh gonorrhœa is almost certain to be followed by a return of the disease; and it should be pointed out to him that each successive attack is apt to be more severe, even more refractory to treatment, and more likely to cause permanent disablement.

Pathology.—Various hypotheses have been propounded to explain the relationship of the pseudo-rheumatic troubles to the urethral discharge; these may be summed up as follows:—

i. That the gonorrhœal infection awakens a latent rheumatic tendency, provoking an attack of true rheumatism; ii. That the arthritis is of nervous origin, and is due to irritation of the urethral mucous membrane; iii. That the phenomena are of pyæmic origin; iv. That the so-called gonorrhœal rheumatism is due to a specific systemic infection by the gonorrhœal virus.

i. The rheumatic hypothesis hardly need be combated at the present day, for there is no longer any widespread doubt of the distinct nature of gonorrhœal rheumatism. On the other hand, cases are occasionally met with in which to all appearance a gonorrhœa acts as the immediate cause of an attack of acute rheumatism of the ordinary type.

ii. The second hypothesis, which has been ably maintained by Dr. Ord and others, does not afford any explanation of the abarticular phenomena of the disease; it is, moreover, opposed by the fact that the articular lesions themselves are not of the same kind as these joint lesions, such as the arthritis of tabes or of syringo-myelia, which are looked upon with the best reason as having a nervous origin, and which exhibit a far closer resemblance to those of rheumatoid arthritis. Nor can the origin of the lesions in urethral irritation be maintained if those examples be accepted in which gonorrhœal rheumatism has followed conjunctival inoculation.

iii. and iv. The rarity of suppuration of the affected joints and the other characters of the arthritis serve to distinguish gonorrhœal rheumatism from an ordinary pyæmia; and the view of the disease as the result of the generalisation of the specific poison is that which can be supported by the most cogent arguments.

We have seen that the symptom-complex here presented is a peculiar one and unlike that of any other disease—a point to which Fournier

was the first clearly to direct attention; the malady has the character of a definite specific disorder, comparable to true rheumatism, but essentially different from it.

The generalisation of an infective disease which in most instances remains localised may be due either to the dissemination of the specific organism, or to systemic poisoning by the chemical products of its growth. The evidence which is forthcoming points to the former as the correct interpretation in this instance. It is easier to explain upon this view the fact that of the many sufferers from gonorrhœa, a few only present gonorrhœal rheumatism; and, since Petrone and Kammerer first investigated the subject, many observers have sought for the gonococcus in the synovial fluid from the affected joints and in the vegetations situated upon the endocardium in cases in which death has resulted from ulcerative endocarditis. The evidence which they adduce is of varying degrees of cogency, but one by one the special properties of the gonococcus have been recognised in the organisms obtained from these situations. It must now be regarded as an established fact that, in some cases at any rate, there are present in the fluid obtained from the joints both of infants and adults, in the blood, in the pleural exudation and the cardiac vegetations, diplococci which present the characteristic shape of the gonococcus; they lie for the most part within the leucocytes, which are decolorised when Gram's method of staining is employed, and they grow readily upon a mixture of human blood-serum and agar-agar, whereas they refuse to grow upon any ordinary culture medium.

Indeed, after reading such accounts as those of Leyden, Bordoni-Uffreduzzi, and Thayer and Blumer, one can no longer doubt the dependence of the above-mentioned lesions upon the dissemination of the gonococcus. Bordoni-Uffreduzzi even succeeded, by means of subcultures of the organism obtained from an affected joint, in producing gonorrhœa in a healthy man who offered himself as the subject of the experiment. On the other hand, it would appear that in certain cases, as in one recorded by Ely, ulcerative endocarditis occurring in association with gonorrhœa may owe its origin to other micro-organisms, and that mixed and secondary infections play as important a part in this as in other infective diseases. Even in the inflamed joints the finding of the gonococcus has not been so frequent as might be expected, and there is reason to think that it may only be present there during the earliest stages of the arthritis.

There are some grounds for the belief that local ulcerations favour the dissemination of the virus, and that the comparative immunity of women from gonorrhœal rheumatism is due to the differences in the length and structure of the urethra in the two sexes.

Treatment.—The first object in the treatment of gonorrhœal rheumatism is the cure of the urethral discharge in which it has its origin; with this end in view the ordinary remedies should be applied both by the mouth and in the form of injections.

With regard to the medicinal treatment of the disease little can be

said that is at all encouraging. The salicylic drugs and colchicum alike are without appreciable effect upon the articular lesions, and lapse of time is the chief means of cure. Iodide of potassium is the drug which is most relied upon, and I am in the habit of giving a mixture containing quinine with an alkali, together with the iodide, and I believe that this line of treatment is by no means without good effect. If the pain be severe, opium may be administered.

In severe cases the inflamed joint may need a splint, and the application of an ice-bag usually affords relief. When the effusion into the articular cavity is very abundant, paracentesis may be called for; and in the rare instances in which a joint suppurates, further surgical interference will of course be required.

The great tendency to the formation of fibrous adhesions must never be lost sight of; if the joint be put upon a splint, care must be taken that, if ankylosis should result, the limb may be fixed in as advantageous a position as possible. Although complete recovery of the affected joints is the rule, at least in the milder cases, some degree of stiffness often remains, which calls for passive movement under an anæsthetic; the minor degrees of residual pain and stiffness may often be got rid of by a course at some mineral water resort where external treatment is carried out, such as Bath, Harrogate, or Woodhall, in our own country. It is, of course, only from the external application of mineral waters that any results can be looked for.

ARCHIBALD E. GARROD.

REFERENCES

1. BORDONI-UFFREDUZZI. *Deutsche med. Wochenschr.* xx. p. 484; 1894.—2. DEUTSCHMANN. *Archiv für Ophthalmologie*, xxxvi. Abth. 1, p. 109; 1890.—3. ELY. *Med. Record N. Y.* xxxv. p. 287; 1889.—4. FAITOUT. "La Pleuresie blenorrhagique," *Arch. gén. de méd.* iv. p. 404; 1895.—5. FOURNIER, A. *Dict. encyclop. des Sciences méd.*, article "Blenorrhagie."—6. GOWERS. *Clinical Journal*, iv. p. 113; 1893.—7. GLUZINSKI (abstract). *Brit. Med. Journ.* i. p. 1084; 1889.—8. HOECK. *Wiener klin. Wochenschr.* vi. p. 736; 1893.—9. KAMMERER. *Centralb. f. Chirurgie*, xi. p. 49; 1884.—10. LAPERSONNE. *Arthrites Infectieuses*, p. 58, 1886.—11. LEYDEN (Myelitis). *Zeitschr. f. klin. Med.* xxi. p. 607; 1892.—12. *Idem* (Endocarditis). *Deutsche med. Wochenschr.* xix. p. 909; 1893.—13. LINDEMANN. *Beitrage z. Augenheilkunde*, I. Hft. v. p. 30; 1892.—14. LUCAS, CLEMENT. *Brit. Med. Journal*, ii. p. 57; 1885.—15. MACDONNELL, R. L. *American Journ. Med. Sci.* ci. p. 1; 1891.—16. PETRONE. *Rivista Clinica di Bologna*, p. 94; 1883.—17. PHILLIPS, S. *Brit. Med. Jour.* ii. p. 1215; 1886.—18. PONCET. *Archives d'ophthalmologie*, i. p. 213; 1881.—19. THAYER and BLUMER. *Johns Hopkins Hospital Bulletin*, vii. p. 57; 1896 (containing a summary of earlier literature).—20. See also a summary of our knowledge of the gonococcus, and a bibliography of the subject, by Neisser u. Schaffer in *Ergebnisse de allgem. Aetiologie, etc.*, Lubarsch and Ostertage, i. p. 503; 1896.

A. E. G.

RHEUMATOID ARTHRITIS [1]

Definition. — A progressive degeneration of the joints of a special kind, accompanied by atrophy of some structures and hypertrophy of others. The course of the disease is variable, but is ordinarily towards irrevocable damage and ruin of the structures involved.

The name Rheumatoid Arthritis was proposed by Sir A. Garrod in 1858. It is short, distinctive, and has taken firm root in this country. There are cogent reasons against disturbing it. It is not necessary to enumerate the other names which at divers times have competed with it; but we must note that the name osteoarthritis is officially recognised by the Royal College of Physicians of London.

The pathological position of rheumatoid arthritis will be better seen when a comparison is made with other forms of arthritis, as follows:—

(i.) Traumatic arthritis indicates the immediate or remote effects of an injury to a joint.

(ii.) Diathetic arthritis is a manifestation of gout, rheumatism, tubercle, scurvy, and the like.

(iii.) Enthetic arthritis may occur in the course of scarlet and enteric fevers, dysentery, syphilis, gonorrhœa. This name includes all kinds of septic arthritis, whether arising from puerperal or other like conditions. The word rheumatism should never be associated with enthetic arthritis.

(iv.) Neural arthritis comprises all joint diseases which are the sequel of central or peripheral nerve-lesion. The word neur-arthropathy conveniently represents this class of arthritic cases. It is logically helpful, for it introduces a new order of ideas; and it rescues us from those primitive conceptions of arthritis which were always inadequate and are sometimes misleading. Thus rheumatoid arthropathy would have been a better name for rheumatoid arthritis had it been adopted in 1858. In the border-land or neutral territory of arthritis physicians and surgeons contend in honourable emulation. When we have delivered ourselves from the traditions that (*a*) all arthritis, as its terminology no doubt indicates, is inflammation, and (*b*) that arthritis nearly always means something rheumatic or gouty, we shall hold a more lucid creed. The word rheumatism was built upon a narrow theory. As the Greek root of it signifies, it denotes "a humour floating in the blood, causing disease"; and we shall see by and by that the name rheumatoid arthritis, defective as it is, preserves an important idea.

Pathology. — There are two main forms of rheumatoid arthritis: (A) the primary, and (B) the secondary.

[1] Before Dr. Kent Spender had completed or revised his essay he was compelled by a heavy affliction to relinquish his task. Dr. Garrod most kindly undertook to revise and finish the work. Dr. Garrod's contributions are indicated by signature or initials.

A. *Primary form.*—The main cause of this form is general weakness—that weakness which follows mental wear and worry, loss of blood, mucous fluxes, unfavourable hygiene, prolonged lactation, or too frequent child-bearing. Many structures and functions suffer. Why, under circumstances apparently alike, the joints are affected in some persons and not in others cannot be explained. The lesion of a joint which we call "rheumatoid" may perhaps be determined by a previous hurt or the chronic strain of excessive use. Joints which have been sharply and continuously engaged in the labour of daily life are liable to fall into degeneration or dystrophy. Over-exertion is not a necessary antecedent, nor even a common one, but whatsoever weakens a joint renders it more prone to a specific deterioration. In one patient, perhaps, there has been a habitual menorrhagia, with intermittent leucorrhœa, from early womanhood; or the menstrual function has been disordered, or some anxiety or a neuralgia has undermined sleep and made restless days. The importance of uterine disorders as causes of rheumatoid arthritis has been urged by Dr. Ord; but the facts have been differently interpreted by other observers.

A family heritage of tubercle is an important factor in some cases. Two or three brothers and sisters, perhaps, show signs of tuberculous disease, and another member of the family may fall into this rheumatoid state. What pathological link binds the tubercle with the arthritis? There is no reason to suppose that this form of articular disease is strumous. The ætiology and clinical course of a rheumatoid joint have nothing in common with struma, though both proclivities may be traceable to analogous causes. Not infrequently a connection exists between influenza and rheumatoid arthritis. The severe epidemics of influenza during the last few years have left their effects in various ways, and this is one of the gravest of them. Patients will voluntarily describe the sequence as a fact about which there can be no doubt. The influenza departs, but an ominous weakness is left behind. Arthritis does not necessarily belong to influenza in any shape, except (as Dr. Sansom has said) as an acute accident of an acute disease. We are confronted with a like constitutional problem to that of tuberculosis; but, in addition, there is the grave poisoning of nerve-centres and the disturbed nutrition which is a result of it.

In all these cases the term primary rheumatoid arthritis is justified by the fact that a state of the system and of the general health had been brought about which is favourable to degenerative processes; of these rheumatoid arthritis is one.

[It is necessary to mention here that a new direction has recently been given to the study of the pathology of the disease under consideration by bacterial investigation of the fluid from the diseased joints. A few years ago Max Shüller described bacilli obtained from such fluid, which had the form of short thick rods, with polar bodies which appeared bright or dark according to the character of the illumination. These bacilli stained readily with carbol-fuchsin, not quite so readily with

methyl violet, and more readily with methylene blue, or Bismarck brown. When cultures of these bacilli were injected into the joints of animals, changes were observed similar to those met with in rheumatoid joints; suppuration never resulted, and the lesions differed from those seen when other bacteria were similarly introduced. Somewhat similar results have been independently obtained by Ballantyne, Wohlmann and Blaxall. After a prolonged and fruitless search in the blood and synovial fluid of sufferers from rheumatoid arthritis, the two former observers ultimately discovered a bacillus which they regard as the cause of the disease. This organism is described as a short bacillus, of dumb-bell shape, which is stained by fuchsin or methylene blue, but not by Gram's method. The poles take the stain more readily than the intervening portion, and hence the appearance of a diplo-bacillus is apt to be assumed. Blaxall confirmed these observations, and eventually succeeded in cultivating the organism.

It was met with in the synovial fluid in twenty-four out of a series of twenty-five cases examined, and in a few instances was found in the blood also. In cases of synovitis of other kinds it was not met with, although organisms resembling it in morphological features were sometimes found.

Still more recently Chauffard and Ramond have called attention to the occasional enlargement of lymphatic glands in the neighbourhood of the diseased joints, the degree of enlargement varying with changes in the condition of the joints. In scrapings from excised lymphatic glands and in the synovial fluid in such cases, they found a slender diplo-bacillus which stained readily, but they were unable to cultivate it in nutrient media.

Should further investigations confirm these results, and if it be shown that some at any rate of the cases included under the name of rheumatoid arthritis actually owe their origin to a specific organism, the prevalent views as to the pathology of this disease will have to be largely revised.—A. E. G.]

B. *Secondary form.*—This is the special lesion which occurs after rheumatic fever—a sequence which justifies the adjective rheumatoid. The condition which is called "chronic rheumatism" often forms an intermediate stage, though not a necessary one: it may remain as such, or darken gradually into the rheumatoid disease; but the two ought never to be confounded. A man or a woman in early middle life may have two or three attacks of acute rheumatism close together: before there is a recovery from one, another begins; wave succeeds wave, and almost overtakes it; no one pyrexial period is intense or much prolonged. Periods of comparative quiescence occur, during which the patient, though somewhat crippled, stumbles about with convalescent energy; he never quite emerges, however, from the condition which, up to a certain point, is properly called "rheumatic." Concerning such a sufferer our unceasing anxiety is whether he is on the way to rheumatoid arthritis. The two groups of clinical phenomena appear to coalesce: the latter may seem but a

postscript of the former, continuous in time and quality; but potentially they are distinct. Pyrexia has gone, but the pulse remains quick, and even quicker than before; the sweating proper to rheumatic fever has passed away, but another kind of sweating has begun. A new series of clinical phenomena arises, some of which, such as pain and enfeeblement of the joints, are a mere continuation of the old; others again are the beginning of a new order of things.

Here then we see not only something chronic in the room of something acute, but a slow process of arthritic disease in another kind. The steps of the sequence are seen only by the practised eye. So far as the joints are concerned, the transition is not well marked; what was swollen and painful is swollen and painful still. The lesson is not always easy to read; but a study of the concurrent facts may make it clearer, and these facts will be discussed presently.

It would be rash to assert that there is no such thing as acute rheumatoid arthritis. But the state so described is usually a pyrexial rheumatic forerunner, which, in predisposed persons, leads on to the rheumatoid state proper. In the great majority of persons there is no such predisposition. Rheumatism comes and goes; it leaves some trace of damage, perhaps, but one which for the most part is quite remediable. Residual adhesions and stiffness represent the "chronic rheumatic arthritis" of the nosologist—a state which must be marked off in a decisive manner from rheumatoid lesions. However the boundary lines may be blurred now and then by complexity of temperament, or by the blending of diathetic taints, the distinction must be firmly maintained; for whatsoever rheumatoid arthritis may prove to be, it is assuredly not chronic rheumatic arthritis. [See p. 59.]

Until lately it was customary to speak of rheumatoid arthritis as a sequel of gout. But "uratic arthritis," whatever it may be, is to be held responsible for all the articular deformities and distortions of gout. Until we know that gout is not really adequate to produce them, no other agency should be brought into the explanation. Similarity is not necessarily identity.

It is acknowledged that there is such a malady as "rheumatic gout"; but the name is used in a sense quite alien to that of the old nomenclature. Often have I seen among elderly folk (chiefly men) in the Bath Mineral Water Hospital a complex disorganisation of the knee-joints, preceded by a long and weary history of rheumatism and acquired gout. In them the operation of a composite diathesis like gout and rheumatism together produces an arthritis of a definite kind; and to this the name "rheumatic gout" must be confined.

Charcot arranged all cases of rheumatoid arthritis in three divisions: (*a*) Heberden's nodes; (*b*) The general progressive form; (*c*) The partial or monarticular form.

(*a*) The *nodosa chiragra* of Horace is so impressed on the mind of the laity, that a nodular finger (especially in its terminal phalanx) is assumed to denote gout beyond all question. Both Todd and Begbie associated

the two as cause and effect. The true node of Heberden is often a sign of a merely local degeneration, especially in elderly people. In people of middle age the existence of these nodules has a pathological meaning; it helps to determine the nature of an arthritis which might otherwise be doubtful. "They may be regarded," writes Dr. Archibald Garrod, "as constituting the slightest and most chronic manifestation of rheumatoid arthritis." Similar nodules are seen now and then on the dorsal aspect of the middle joints of the fingers. The nodules themselves are due to an "osteophytic enlargement" of existing structures; they are much more common in women than in men, and Dr. Osler says that the subjects of these nodules rarely suffer in the larger joints.

Heberden's nodes represent a partial and lenient manifestation of rheumatoid arthritis. The disease becomes quiescent (or even extinct) after a certain advance; but the marks remain. Such a person, after a time, may become virtually non-rheumatoid, and may acquire gout or any other malady having no causal connection with the nodules. These are landmarks of "battles long ago."

(*b*) The general progressive or polyarticular form is Charcot's second division. Many writers testify that rheumatoid lesions generally begin in the hands or the feet, and do not appear elsewhere. The hands are often affected first; and very seldom do they escape altogether. This order of invasion obeys what has been called the "centripetal law." To this rule there are many exceptions; and we must admit that the "natural history" of a disease may be thwarted or helped by external circumstances. Let the problem be studied as it may be presented at any time in the Bath Mineral Water Hospital. The joints which have been most actively engaged in the special craft of the patient, whether this be husbandry or carpentry, needlework or washing, are usually the first to show signs of arthritic degeneration. Nor can the daily toil be stopped: the mangle, the spade, or the needle must go drudging on; and too infrequent is the rest so urgently needed. Organs which are overworked exhibit the first signs of decay. The feet may be primarily affected if the occupation of the sufferer has demanded excessive walking or standing.

But the capacities of the human hand are so many and varied, that unfair stress may be laid upon it even by patients who have not been engaged in manual labour. If we imagine that the petty joints of wrist and instep, and fingers and toes, represent in each case a big joint split into segments for a special function, it becomes evident that all the risks of arthritic disease are arithmetically multiplied. The feet often enjoy comparative repose while, in ministering to the business of life, the hands may never be idle. And in that restless energy which always goes on except during sleep physical wear and tear is an inevitable result.

The peripheral incidence of rheumatoid arthritis must be accepted, therefore, with some qualification; it may be true, but the facts may be

otherwise interpreted. The high endowments of the hand imply a correspondingly high level of nervous organisation. The complex motor and sensory apparatus of the upper limbs meets a physiological need; but this very complexity may make it more vulnerable in disease. The force of the argument drawn from neural considerations is so weighty that there is no room for the curious surgical doctrine that the articular lesions in rheumatoid arthritis are the results of the mere contrition of opposing surfaces.

Charcot said that the lesions tend to advance up the limbs towards the trunk. But although the wrists are generally affected after the fingers, and the ankles after the feet, not seldom are the carpal and tarsal articulations affected first. The elbow often escapes, whilst the shoulder is involved. In the lower limbs the knees seldom escape; but, in this form of the disease in comparatively young people, the hip-joints are not often affected.

The temporo-maxillary articulation now and then becomes stiff and painful at an early stage. It is attacked by rheumatoid disease in about a quarter of the total number of cases. It is an important diagnostic sign; but there is at least one form of septic infection which may attack this joint. [See p. 66.]

The symmetry of rheumatoid arthritis has been long observed. Occasionally, however, there is a hemiplegic distribution, not only of the arthritic phenomena, but of all the companion symptoms.

At a later period of the disease the articulations of the spine may become engaged. The cervical vertebræ are usually attacked first; a difficulty is felt in rotation and nutation; and often the discomfort is referred to muscles rather than to bones. The dorsal and lumbar vertebræ may be crippled next, so that nearly the whole length of the spine is transformed into a rigid column. The body is twisted and shortened, and moves in one piece; and in the recumbent posture the muscular difficulties are great: in a number of cases, however, the disease does not go beyond the cervical spine, or is checked by appropriate treatment.

In this form of disease the large preponderance of patients is among women.

(*c*) The partial or monarticular form of rheumatoid arthritis is Charcot's third division. It is seen in the knee, hip, spinal column, and shoulder. The joints which are nearest the trunk suffer the most; and by older writers the term "hip-ache" was applied to all pains in that part, if the nature of the pain, whether due to sciatica or disease in the hip-joint, could not be nicely discriminated. But it was recognised that men suffer much more than women; and, as men are more exposed to injury and to the physical danger arising from occupation, so this form of rheumatoid arthritis came to wear a surgical aspect. A very slight injury may give the first impulse to senile change. But what a relative word *senility* is! In medicine (says Prof. Clifford Allbutt) we do not count the ages of people by the revolutions of the earth around

the sun, but we measure them by the involutions of their own morbid processes. Observe that the so-called monarticular lesion tends to spread and to become multiple; rheumatoid hips are apt to be followed by rheumatoid knees, and infirmity in the elbow may follow infirmity in the shoulder. Very rarely are the hips crippled without engagement of the neighbouring lumbar and even of the lower dorsal vertebræ. But a polyarticular lesion may become grafted upon the other, when the patient is "young in years" though "old in hours." An active person between seventy and eighty years of age exhibits now and then a rheumatoid hand or foot which has appeared as a sequel of arthritic trouble in shoulder or hip; either of which may have been monarticular for some time previously.

The most strictly localised form is that which occurs after an injury. The prematurely senile type is distinguishable by other symptoms which in themselves indicate premature senility—such as degeneration of the hair, teeth and arteries.

Morbid anatomy.—It were a purely academic discussion to inquire whether the morbid changes in rheumatoid arthritis are primarily inflammatory or primarily degenerative. Probably both processes are concerned in the change. The nature of the inflammation determines the resultant hyperplasia. Rheumatoid disease is a near neighbour to tuberculosis; and when a proclivity to tubercle exists in a family, we may expect that any rheumatic member of that family may become rheumatoid.

The disease has been called a panarthritis, because it involves all the parts of a joint—cartilage, bone and synovial membrane. Rheumatoid destruction is contagious in its potency; and the total result is much the same wherever the ruin may begin. The nutrition of all the constituent parts of a joint is affected, causing proliferation first and absorption afterwards. The articular ends of the bones are thickened and flattened out, with protuberant margins which overlap the diaphysis like projecting lips. The matrix of the articular cartilage becomes split up into delicate fibrillæ, whilst the capsules disappear. At this stage the cartilage has a velvety appearance which is well seen in museum specimens of rheumatoid joints. Later, the fibrillated cartilage is removed, leaving the surface of the bone exposed; and such denudation takes place earliest at the points of maximum pressure. Mutual friction of the ends of bones during the movement of an articulation gives them a smooth and lustrous surface, traversed by fine striæ. Bones thus affected are said to be eburnated, and the superficial layer of the bone is found to have acquired a greatly increased density.

There is a marked contrast between the results according as the receptive cavity for the head of a bone approaches the socket type of joint (as in the hip), or as it is very shallow in its concavity. In the former case, by the process of grinding, the deep concavity may be so enlarged that the ball of the femur will slip out of its place into a new socket thus made for it. A shallow articular surface, on the other hand, like the

glenoid cavity of the scapula, may be so narrowed and deepened by stout marginal protuberances, that the mobility of the head of the humerus is much impaired. The morbid changes are confined to the epiphyses.

The synovial membrane is nearly always thickened. There is an increased amount of synovia, which is cloudy, thin, and mixed with ground-down particles of cartilage. Fatty degeneration affects ligaments and interarticular cartilages. The capsule may be ossified by thick plates of bone (called *addimentary bones* by Dr. R. Adams); and all parts about a joint—periosteum, tendons, muscles—may participate in the slow inflammation until the ends of the bones are covered with more ossifying masses. Some osteophytes are flat and roundish, "looking like a fluid which had poured out and stiffened while flowing"; others are shaped like pointed stalactites. In the midst of the subserous cellular tissue detached points of bone are apt to form, which for a long time remain as isolated rounded pieces; subsequently, however, they may unite with other bony masses.

Chronic inflammation of synovial membrane is usually the first event in time; disease of cartilage is the first in importance. The progressive ossification of the overlappings of cartilage leads to the formation of the nodular protuberances which characterise the disease.

It is evident that several factors contribute to the swelling of joints, and to their deviation from normal shape. The great thickening of capsular ligaments is increased by osteophytes, and, owing to the retraction of muscles, these look larger. The tendons of muscles are stretched and thinned.

Periarticular bony formations may cause entire dislocation of a joint, and force it into an abnormal half-luxated position; or they may render it immovable. But stiffening and almost complete immobility do not make true ankylosis; although fibrous adhesions and periarticular thickening simulate the dense cicatricial tissue which binds together the ends of bones in the distinct chronic rheumatic arthritis (see p. 59). In the rheumatoid lesion the bones are not joined into a rigid piece (except in the spinal column); it is the block of surrounding alien masses which impedes or prevents movement.

Interarticular cartilages and ligaments are more or less destroyed.

Now and then a curious inversion occurs in the physiological anatomy of a joint. Where a cavity should be, a "convex boss" appears, which may actually fit into an excavated cup replacing the ball of the adapted bone.

The muscles usually show a decided atrophy, and neuritis of the peripheral nerves has been found. The senile changes of the localised rheumatoid arthritis are practically identical with those which characterise the progressive form, but they are milder in degree. All are phases of a dystrophic manifestation.

Clinical History.—A certain identity runs through the clinical forms of rheumatoid arthritis whatever its causation. Concerning any given case of which the history is veiled from us, it would be difficult to say

from its essential or accidental symptoms whether the disease were primary or secondary. And even the history itself may be equivocal. A remote attack of pyrexial rheumatism may have nothing to do with a recent attack of rheumatoid degeneration; for during the years which separated the two events other depressing forces may have come into play, impoverishing the blood and shaking the nerve-centres. A general similarity in the two chief types of rheumatoid arthritis includes a vast number of petty variations. When there are no known or apparent rheumatic premonitions a case should be considered from the neural standpoint.

Modes of onset.—(*a*) A woman in delicate health goes to bed, and is awoke from sound sleep in the middle of the night by paroxysmal pain in one joint, commonly the shoulder or the elbow. Movement increases the pain. There is no sign of pyrexia, and the patient is not "ill" in a medical sense of the word. (*b*) After slighter bed-time warnings a patient's sleep is broken by a burning pain in one hand. The whole limb is thrown outside the bedclothes, and tossed about for cool air. (*c*) Early one day a person feels a sudden weakness or tiredness in the right hand and a tenderness along the inner border of the carpus. The weak hand is grasped by the other, or a handkerchief is wrapped around the wrist. (*d*) After a long and fatiguing walk a woman feels a "chill" around her cervical spine and muscles; within a few days rotation of the upper vertebræ is seriously impaired. The arms cannot be uplifted; in a short time neuralgic pangs stream along the limbs, a prelude to the arthritis which sooner or later comes on. (*e*) At the end of a prolonged lactation pain and swelling occur in one joint. Their significance is not immediately recognised. (*f*) A person, not consciously out of health, finds a sudden weakness in the hands: they drop small objects; the fingers cannot pick up little things; the complaint is that the hand will not "hold," and that the fingers are "going to be paralysed." Along with this motor weakness there may be sensory aberrations, variously described as "burning," "scalding," or "scraping." Not infrequently there are evanescent blanchings and blushings, or again a permanently red or purple tint of skin. Small cutaneous areas are sometimes decidedly chilled. (*g*) Has not the ankle-joint been strained?—is a question often put to a sufferer whose foot conveys the first warning of swelling and weakness. And for a while, perhaps for months, the pity and the nursing are bestowed in the wrong way, and the right kind of help is not suggested until arthritic mischief has become a settled fact. (*h*) Now and then a rheumatic patient complains of severe "muscular pains" in the neighbourhood of a large joint, coming and going without apparent cause. A severe prophetic note of the coming storm is a pain in the ball of the thumb; and hardly less common is a sharp pang on the inner side of the wrist. Both these forms of neuralgia are very characteristic of incipient rheumatoid change. What we imagine to be a pure sciatica is now and then a rheumatoid invasion of the tarsal articulations, knee, or hip.

This sketch of the sundry modes of beginning of rheumatoid arthritis by no means exhausts them. No medical trouble is more insidious in its approach or more subterranean in its quiet journey. The simple statement that a disease may be well on its road before a suspicion of its presence is aroused demonstrates the practical importance of a right understanding of this matter.

Very soon, if the arthritic lesion be of the rheumatoid kind, a dull, slow swelling appears in the menaced joints. The wrist is bulged and tender; swelling is obvious when the hand is held up and viewed laterally. The first two metacarpo-phalangeal articulations are usually the first parts affected on the distal side of the wrist, and sometimes the other knuckles are not touched at all. The interphalangeal joints of the fingers swell, and the enlargement of the first joint beyond the knuckle imparts to the whole finger a spindle shape. Any enlargement of the second interphalangeal joint is masked, perhaps, by Heberden's nodes. Here, then, is the first aggression of the disease in many young adults; nor may it go beyond this stage for months or years. The thumbs escape, or only their carpo-metacarpal articulations are involved. The foot quickly shows sympathy with the hand. Tarsal bones enlarge and become tender; there is synovial effusion among the complex articulations; the elasticity of the plantar arch is spoiled, and even slow walking becomes difficult.

The natural attitude of flexion of the knee and elbow keeps them at ease, and friction between joint-surfaces is lessened. Difficulties arise in the hip or the shoulder either immediately after those of the foot or the hand, or after a considerable interval of time; and the knee is more often affected severely, because in its office it has to bear the weight of the body. But there is no definite law of sequence in the matter.

A limitation of movement and a grating noise when the ends of the bones are rubbed against each other are proofs that the mechanism of a joint has gone wrong. The crackling of periarthritic adhesions gives a peculiar and unmistakable noise; and the restrictions which these adhesions impose upon freedom of rotation may be as abrupt and decisive as if the bones themselves were at fault.

Atrophy of muscles is uncertain in rigidity and degree. This atrophic process is often an early symptom, and synchronous with the arthritic disease; this fact has suggested to many observers that both conditions depend upon a common cause. Sometimes the muscular wasting is distinctly in advance, and is out of all proportion to any mischief in the nearest joints. Along with it there is often a "cutaneous dystrophy"; the skin is glossy and pink, or it is translucently pale and shines like a mirror. The fingers and hands often afford examples of glossy pinkness, and those parts of the limbs which are ordinarily covered with clothing may be waxy white. The skin on the flexure side of the fingers becomes thin and brittle. The nutrition of nails occasionally suffers.

How are we to explain this group of atrophic phenomena? Three hypotheses have been entertained:—

(i.) The atrophy of muscle may be myelopathic, and have a central spinal origin. The argument may be stated thus:—A patient suffering from rheumatoid arthritis with much atrophy of limbs may also present the signs of bulbar paralysis. If there be a lesion in the anterior horns of the spinal cord, especially in the cervico-dorsal region, such a lesion may well extend to the medulla oblongata, where are certain motor nuclei continuous and homologous with the anterior horns. The phenomena of progressive muscular atrophy may thus be added to those of a rheumatoid atrophy of spinal origin, and consist in a rather quick loss of power in the muscles of the shoulders, neck, chest, and also in the intercostals. The tongue cannot be protruded to the usual extent. The action of the masseter and pterygoid muscles becomes feeble. There is an altered expression and a fallen or blank look about the lower part of the face. The speech is probably slow; the respirations are short and panting, and the complexion is blue. These symptoms convey plain evidence of a polio-myelitis at the root of the hypoglossal nerve, of the *portio dura* of the seventh nerve, of the motor root of the fifth nerve, and of the root of the spinal accessory. There is never any positive recovery from this condition; on it marches to its certain end, and death occurs from paralysis of respiratory and cardiac nerves. It is remarkable that the arthritic part of the malady may wane while the bulbar complications steadily increase. The sketch just given is drawn from two cases which were long under my observation.

Besides these graver paralytic symptoms there may be incoördination of the pharyngeal muscles. Not only is the act of swallowing done with hesitation, but now and then it is but partially accomplished. A regurgitant rush of food through the nostrils tells of spasm; the way into the œsophagus is temporarily closed. A "nasal voice" is not uncommon. Such a pharyngeal crisis comes on occasionally in the course of tabes. It is curious that, after a comparative recovery from rheumatoid lesion in joints, a pharyngeal difficulty may come as something quite new; as if the neurosis which is at the root of the rheumatoid disease had not passed away.

The existence of a nerve-centre which presides over joint-nutrition has been postulated by more than one physiologist. The evidence for a central cause of the atrophic phenomena of rheumatoid arthritis was given by Senator nearly twenty years ago. Even if we admit, thus he wrote, that these and other forms of joint-disease occurring in the course of nerve maladies are due partly to injury, we ought to note the very frequent coincidence of joint affections with diseases of the central nervous system. Rheumatoid arthritis may be set agoing by violent emotions and sorrows; it is commonly symmetrical in its progress; it is often associated with neuralgic and tropho-neurotic symptoms, and the muscular wasting is out of proportion to the joint mischief. And who can fail to see an analogy between this arthritis and the arthropathy originating in disease of the spinal cord, as in tabes? I may add that a similar arthropathy has been described as an occasional result of concussion of the spine.

In what is called infantile paralysis (although it may occur at any age) paralysis quickly follows a lesion of the ganglion cells in the spinal cord. The muscles affected are flaccid and toneless from the first. By the second week the reaction of degeneration is present. Reflex action from the skin and myotatic irritability are abolished. In progressive muscular atrophy, however, faradic reaction may remain until all the muscle is wasted away. The flexor muscles are usually affected before the extensors, and the wasting in the legs is much less common than in the arms. In a case of rheumatoid paresis the electric irritability of the atrophied muscles is usually normal. Generally there is a distinct increase in myotatic irritability; if the thigh muscles are affected the knee-jerk is excessive, and sometimes a rectus clonus can be obtained. A foot clonus may be present when the ankle-joint is affected. In some cases there is a slight rigidity of the affected muscles, which are anything but toneless; and those which waste are chiefly the extensors of the affected joints. It must be confessed that it is difficult to reconcile all the phenomena on the hypothesis of a spinal lesion, and no such lesion has ever been discovered.

Examples of rheumatoid disease are not uncommon in which we see a debility that approaches a spinal paralysis, but without conspicuous atrophy. And the dawn of trouble is betrayed sometimes by slight motor failure; little objects (like a cup or a jug) slip out of the hand unawares, or the manual grasp unconsciously becomes feeble.

(ii.) Pitres and Vaillard believe that there is a constant relation between the presence of neuritis and the development of trophic changes in the muscles and skin. Dr. A. Garrod admits that the "peripheral element" will have to be considered; and the problem will be how far the lesions of peripheral nerves determine "irregularities in the relation of myotatic irritability to the muscular atrophy, the occurrence of numbness and tingling and cutaneous dystrophy, and even changes in the joints themselves." Dr. T. Oliver shows that in lead poisoning the lesions are both central and peripheral.

The ultimate appeal must be to pathological anatomy. Partly from the paucity of material, and partly from the disturbing agency of intercurrent disease, the results of examination after death in rheumatoid arthritis are at present uncertain and even contradictory.

(iii.) The muscular atrophy of rheumatoid arthritis has been called "arthritic," in the sense that when a joint ceases to be used the vital machinery which moves it tends to deteriorate. Atrophy is said to follow disease of the joints in nearly all cases. But it is certain that dystrophy of muscles often goes on step by step with that of joints, or even precedes the latter. That the wasting of muscle is often independent of the mischief in neighbouring joints is proved by the fact that when rheumatoid lesion in the joints is unilateral muscular wasting and weakness may be as severe on the sound side as on the other. I have a young gentleman now (March 1895) under my care, in whom there is spurious ankylosis of the right elbow; the muscles of the upper arm and

forearm have suffered accordingly, but the whole of the left upper limb has also withered nearly as much, although all the joints are freely movable.

The localised form of rheumatoid change, being a dystrophy arising from injury or similar damage, is that which is most apt to be followed by muscular wasting as a natural sequence. There is a great resemblance to an ordinary surgical malady, with its reflex results.

We cannot yet go much beyond the provisional hypothesis that all rheumatoid atrophy represents a neuropathic or myopathic disturbance of nutrition, and that the nature of this disturbance may be practically the same although proceeding from various causes.

Other behaviours of muscle in this disease must be noted. Cramp and intermittent spasm may last for hours at a time and cause acute pain. If the upper segment of the trapezius muscle be irritated through extension of a vagal storm to the spinal accessory, a paroxysmal torticollis is set up which causes the head to rotate and to become fixed. Tremor seizes a rheumatoid limb now and then, beginning nearly at the same time as the arthritic lesion. The spasm is of the type of paralysis agitans; and a shaking limb may be thermometrically warmer than a quiet limb on the other side. Athetotic movements are not uncommon in the hands. Increased tonicity of the muscles is the cause of the grotesque distortions of the fingers and toes; and the deformities so produced "become in time permanent and irreducible, owing to changes in the wasted muscles which lead to permanent shortening." The exact nature of those deformities is determined by the contraction of muscles or by their wasting, and by the predominance of one or the other condition.

[The name Arthritis Deformans, by which the disease under consideration is generally known in Germany and in America, calls attention to one of its most characteristic, and at the same time one of its most distressing features, namely, the conspicuous deformity and crippling which it tends to produce. Several distinct factors enter into the production of the deformities. In the younger sufferers the fusiform swelling of the joints is only in small part due to actual osteophytic outgrowths, at least in the earlier stages of the disease; the enlargement being chiefly due to thickening of the synovial membranes and other soft structures around the joint. In older patients, on the other hand, the outgrowths of bone are often the main cause of the deformity, which in such cases is apt to begin more abruptly in the immediate neighbourhood of the affected joints. The nodes of Heberden afford an excellent example of this, as they consist almost entirely of osteophytic outgrowths, although their size may be materially increased by the presence of translucent hernial bulgings of the synovial capsules.

Bursal swellings are frequently met with in the neighbourhood of the diseased joints, and are most apt to be conspicuously present on the dorsal aspects of the wrists. Enormous cysts have occasionally been met with in the thigh in connection with rheumatoid arthritis of the hip-joint.

The last and perhaps the most important class of deformities includes those resulting from the spasm of the atrophied muscles. Flexion of the limbs at the knees or elbows is often present, and in extreme cases various grotesque deformities of the hands and feet may be present.

The hand deformities resemble those produced by long-standing lesions of nervous origin, such as anterior polio-myelitis. When contraction of the interossei produces flexion of the metacarpo-phalangeal joints, the first interphalangeal tends to become hyperextended, and there is a compensatory flexion of the terminal joints. When, on the other hand, there is hyperextension of the metacarpo-phalangeal there is a reversal of the above-described attitudes of the interphalangeal joints.

The deflexion of the fingers to the ulnar side, which is so frequently seen in rheumatoid arthritis, appears on the other hand to be intimately associated with past or present disease of the metacarpo-phalangeal joints. It is sometimes seen in its most extreme form in patients who have passed through several attacks of rheumatic fever, apparently attended with relaxation of the ligaments; but so far as I am aware it is never met with as a result of the chronic spasm due to primary disease of the nervous system. That it is not a result of simple spasm acting upon healthy joint structures is further shown by the fact, that although in extreme cases the extensor tendons may slip from their grooves in flexion, such slipping of the tendons is by no means constant.

Deformities of muscular origin may be established at a comparatively early stage of the disease; and they are frequently more marked in cases in which the enlargement of the joints is comparatively insignificant. In the course of time the limbs or hands tend to become fixed in the vicious positions which they have assumed, and the deformities are no longer capable of reduction.

The monarticular form of rheumatoid arthritis does not, as a rule, produce much deformity; but when the hip-joint is affected there may be conspicuous shortening due to absorption of the head of the femur.

In the earlier stages of the disease, when it affects many joints, such crippling as results is, as a rule, mainly due to the limitation of movement imposed by pain; but in the later stages the patients may be rendered completely helpless, partly on account of the restriction of movement which is caused by the locking of osteophytes, and partly as the result of muscular contractures. Seeing that in its more inveterate forms rheumatoid arthritis may successively implicate almost every joint in the body, including the temporo-maxillary articulation, the condition of the patient may become a deplorable one. Fortunately, however, such extreme cases are comparatively rare.—A. E. G.]

In all forms of rheumatoid arthritis the grip of the hands should be regularly measured. The use of the dynamometer is not without its fallacies, and it needs to be corrected by the changing conditions of daily health; but no other test is so easy of application, or upon the whole more trustworthy. Some experience is necessary for fixing the instru-

ment in the grasp of the hand; the arm should then be raised, and the palmar surface of the hand made to look downwards while the spring is compressed. In secondary rheumatoid arthritis (that is, after long sub-pyrexial rheumatism) observation of the dynamometer is particularly valuable. It tells us when the ordinary debility of the diathetic malady is passing into a graver and more lasting paresis. But in the ultimate stages of disease it is clear that bony distortions may prevent the co-ordination of the muscles of the hand; or the muscles may be so wasted that no pressure-test is available.

The changes in the circulation are frequent and important. Many cases of rheumatoid arthritis, rapid and crippling in their march, are characterised almost from the beginning by increased rapidity of the heart's action. The pulse-rate may go up gradually to 90 or 100, and remain so for years. This is tolerably common. In some comparatively rare but very noteworthy cases the pulse may run higher still. Synchronously with the earliest objective signs of arthritic disease the cardiac rate may rise to 110, 115, or 120. Scarcely any variation is observed whether by day or night. I have recorded one case in which a very hard pulse reached the high point of 140. The body is apyrexial, and the purple coldness of the hands is very manifest. There is no sign that the heart is organically affected; no hæmic murmur; no venous engorgement; no local œdema. The heart runs without adequate check; the inhibitory power of the pneumogastric nerve may be partly withdrawn, or partly neutralised by some influence which cannot at present be defined. The tachycardia, once established, never entirely subsides, even though the rheumatoid degeneration may be arrested and recede into quiescence. In the Bath Mineral Water Hospital I once examined all the rheumatoid people (under the age of 60) then under my care in order to discover the frequency of a quickened cardiac action. In 80 per cent of the cases the pulse-rate was found to be more than 90.

We should always examine the arteries of rheumatoid patients who have cardiac irritability. If the blood-pressure be high, and the vessels tortuous, degenerative changes are in progress. Now and then the pulse is distinctly both quick and weak.

It is worth while to study the bare outlines of a case of this kind. A maiden lady, born in or about 1836, was in a state of chronic ill health. A brown pigment gradually appeared on the forehead, which was spotted with little freckles. A quick action of the heart came on during 1880 with a slight polyarticular rheumatoid arthritis. There was no thyroid enlargement. In 1894 the pulse-rate gradually increased to 105. Tuberculosis was suspected twelve or fourteen years ago, and indeed confidently announced. But no visceral disease of any sort has ever occurred. Things remain stationary (1895). What does this curious and persistent neurosis mean?

Between 1884 and 1887 I was struck by the disturbances in the colour of the skin among my patients in the Bath Mineral Water Hospital. The symptom was obvious and even obtrusive, but no

record of any such observations could be found. Concentrated in patches more or less large, the pigment assumes many hues and affects many parts of the body. Across the forehead it spreads as a light bronze smear, or like a dash of chloasma; over the temporal fossa on each side the tint may be deeper. Under the lower eyelids the streak of colour may be very dark, and may shine with a metallic polish. The dominating tints on the face are lemon, orange, and citron; the lustre varies with the angle of reflected light. In people of dark complexion the colour of the face is sometimes so swarthy as to make the patient resemble a mulatto. The white of the eyes stands out in brilliant contrast. In some cases the neck looks as if soaked in walnut dye, beginning at the line at which it is ordinarily covered; and the skin has the appearance of being unwashed.

A bronze shadow can be often detected on the back of the hands. Other parts of the body, quite covered from sun and air, may be darkened. Yellow circles are not uncommon across the finger-joints, and especially around the nails of fingers and toes; and the nails may be streaked with yellow lines. The deep yellow pigment of far-advanced rheumatoid arthritis is a rare form of multiple xanthoma. The plantar surface of the feet becomes rough and horny, and is marked here and there with the brightest yellow stain. A rash then appears on the legs in the form of brownish yellow spots or patches. The spots vary in size from the tiniest specks visible to the naked eye to dots of irregular form from one-eighth to one-fourth of an inch in diameter. In tint the spots vary much; wherever they are most separate the yellow is brightest. A burning and "drawing" sensation is continually felt in the legs and feet. During the night there is extreme heat with great perspiration all over the body. The nails of the fingers and toes gradually separate from their respective phalanges, and are torn or twisted from the matrix by a quantity of dry chalky material. I take this description from a patient who was under my continuous care for more than two years (1884-86).

The ordinary spotty pigment may be thus described:—Each freckle is to the naked eye a mathematically round spot, so well defined that it does not shade off into the neighbouring skin; and it is nearly always of a blackish yellow colour. It is not raised above the surrounding tissues. Its favourite seat is on the forearms; on each arm four to eight spots are commonly to be seen at varying distances from each other, and having no more anatomical relation than the stars of a constellation. Now and then there is a notable spot on the ball of the thumb; here and there a spot may be seen on the neck, and perhaps one on the external ear. They are scattered on the red tinge of the cheeks; and a multitude of specks may strew the forehead, barely visible unless the hair be well thrown back and the light good. Mixed in a random manner with freckles and polished bronze are daubs and brown sprays, as if a shower of diluted Indian ink had been thrown about and allowed to dry. Not infrequent are very black patches, as if solid lunar caustic had been applied to the

parts. In two or three cases I have seen oblong marks of leucoderma, or circumscribed areas of comparatively white skin.

Another colour change is the coming and going of small areas like bruises, which pass through the same phases. Has there been an accidental and trivial hurt? Nothing of the kind can be recalled. We may be attending a patient for a supposed neuralgia of the leg, "gouty" or otherwise. All the classical remedies fail; the case is a riddle; and just when we least expect the riddle to be solved, "odd yellow blotches" appear all down the limb. These "blotches" resolve the problem. They say that the neuralgia is a special symptom of neural arthritis, which is come or is about to come. The bruises are as apt to show on limbs which are not rheumatoid as on those which are so.

In the diagnosis of true rheumatoid pigmentation there are difficulties which are not always small. We have to distinguish all congenital spots and "moles," freckles caused by sun or artificial heat, scars darkened by time, and the natural discolorations of age. There are textural degenerations also which mimic disease of the skin,—a nearly forgotten acne, old pimples, nævuses, and hardened dermoid patches. It is scarcely possible, however, to be misled if we inquire closely into the chronology of the pigmentary stains. People may voluntarily state that the "yellowness" and the "spots" began at the same time as the rheumatoid symptoms; and although hospital patients have far from accurate memories of their ailments, they will sometimes testify that these strange "eruptions" came with their pseudo-rheumatic troubles. However much a rheumatoid patient may improve, both in general health and in the arthritic lesions, these pigmentations never altogether vanish. The trophic processes of the skin seem to have been too profoundly altered to recover.

In every rank of life persons suffering from rheumatoid disease are wont to have very cold hands; the coldness, which is almost cadaveric, extends a little way up the arms. There are three varieties of the rheumatoid cold hand: (*a*) The cold dry hand is always cold and dry; atmospheric conditions do not influence it, but the warmer the atmosphere the greater the contrast between it and the cold hand. (*b*) The cold hand is dry, except on the palm. Here the perspiration streams down the natural furrows, and it feels cold by reason of its contact with cold skin. (*c*) The hand is cold and wet all over; palm and fingers are covered with an abundant dew. The dew is always present whatever may be the hygrometric and thermometric conditions of the external air. The same phenomenon is observed on the feet. We uncover the lower limbs of a rheumatoid patient in bed, and see the large drops sprinkled over the instep, and sometimes a short distance up the leg. The feet are seldom so cold as the hands; mainly, I suppose, because they are always wrapped in clothing. We may dry the feet by artificial means, and keep them as warm as we can, but the perennial flow of moisture is not checked. Sometimes the long trough down the back is a canal of running moisture which continually saturates the clothing.

Another form of sweating is more characteristic of middle-aged women.

In the early hours of the morning all the upper part of the body is bathed with perspiration; and a patient "dares not even put an arm outside the clothes for fear of being chilled." The moisture gradually passes off, and the skin becomes fairly dry again. Whatever the amount of morbid sweating (which is most common in the purely neural forms of rheumatoid arthritis) there is no shivering or evidence of a hot stage.

The various pains have many features of interest. In an early stage of rheumatoid disease the pangs are characteristic, and I have spoken of those which haunt the forearm as having a diagnostic significance. At a later stage the pain is more diffused and less confined to particular nerves. There may be acute and paroxysmal pain in the whole arm, and exploratory pressure over the brachial plexus may be very unpleasant. We search for the morbid changes of rheumatoid arthritis, and if we find them the neuralgia should be regarded as a sure sign of advancing evil. What we may take to be a mere sciatica is now and then a note of early rheumatoid lesion in the tarsal articulations, or perhaps in the knee. More often this specific neuralgia of the lower limb is, so to speak, pain in bulk; the sufferer clasps the thigh all round, and says that pain is in every part of it. It is probable that the lumbo-sacral plexus is swept by currents of pain, the influence of which is felt indifferently through all its branches. Waves of cerebral sympathy are often stirred also by a progressive rheumatoid disease. Intense supra-maxillary neuralgia, subjective perversions of taste and smell, some deafness with a never-ceasing singing in the ears; all or any of these symptoms accompanying the arthritis may lead us to a true diagnosis of its nature, or may strengthen the inferences collected from other quarters.

The clinical symptom of pain points either to neuritis or to irritation of the ganglion on the posterior root of each spinal nerve; and so far it tends to support the central hypothesis of rheumatoid atrophy. But pain and atrophy are not in any definite proportion to each other; no degree of pain at the outset of a case indicates that there will be atrophy later; and the worst atrophy may be unaccompanied by pain. A predominance of either symptom cannot be anticipated from the supposed origin of a case, or from the manner in which it manifests itself. Nor is it easy to unravel the diagnostic entanglements of neuritis, pure neuralgia, or nervous debility. After pressing the nervous threads at different points in their course, and at the places where they emerge from the deep fascia, we may be so far in doubt as to justify the use of an anæsthetic in order to test the condition of a joint which is obviously embarrassed. We may find the range of passive movement so wide as to show that the restraint of active movement arises entirely from the severity of pain, and not from any mechanical bar. In the upper limb I have traced pain especially in the branches of the circumflex nerve over the deltoid muscle, in the external cutaneous branch of the musculo-cutaneous nerve over the supinator longus muscle, and in the dorsal cutaneous branch of the ulnar nerve at the front and back of the wrist respectively. Pain is sometimes

referred to cramp of muscle, and even to the bones themselves. An outbreak of herpes zoster has (within my own experience) been known to occur as a complication of paroxysmal rheumatoid pain.

The extension of disease to the joints of the vertebræ causes irregularities in the vertebral canal, with compression and irritation of the spinal cord, of the nerves arising from it, and of the spinal ganglia. Senator thinks that shooting pains, paralyses, and trophic changes may be attributed to this condition of the spine.

Rheumatoid arthritis is more common in children than is generally supposed. During my official connection of nearly twenty-five years with the Bath Mineral Water Hospital there were admitted between fifteen and twenty children (under the age of 14) suffering from rheumatoid disease. The subject is fully treated in a subsequent article.

At the other end of life it is often difficult to draw a line between the degenerative changes of age and the special lesions of rheumatoid arthritis. A study of a number of people between the ages of 60 and 75 in the hospital just mentioned showed that their alleged rheumatic difficulties might be roughly divided into three groups: (*a*) Those who had no distinct rheumatic history of any kind, but whose joints were suffering from wear and tear: in these cases the occupation of the sufferer determines the place of the first outbreak of the malady; the degeneration is the issue of agencies rather static than dynamic. (*b*) Those who have had several attacks of genuine rheumatism, whose symptoms are those of chronic rheumatic arthritis, and in whom there is no evidence of rheumatoid disease in the strict sense of this word. (*c*) Those who have a monarticular disease, generally of the hip, which we may name as we will. With convenience and accuracy it is called a senile change, of which the remote cause is a strain of ligaments, and not necessarily a fracture of bone. Dr. A. Garrod puts the matter thus: There is some interdependence of the joints and the neighbouring parts, so that injury to the one is followed by a reflex dystrophy of the other. Injury to a joint may be followed by atrophy of neighbouring structures; and decay in a joint follows damage to the soft parts in its neighbourhood. If the disease spread it loses the distinction of being monarticular, and becomes allied to the ordinary rheumatoid type. It is admitted that the morbid changes in the monarticular senile disease are nearly identical with those in the extended and ordinary rheumatoid form.

Periosteal nodes and subcutaneous fibroid nodules are not uncommon in secondary (or post-rheumatic) rheumatoid arthritis, and in that which is apt to follow influenza; but they are much more rare in the primary rheumatoid types. [Occasionally, however, nodules closely resembling those met with in young rheumatic patients are seen in sufferers from rheumatoid arthritis with no obvious rheumatic antecedents, and who have reached a period of life at which true rheumatic nodules are extremely rare. Cases of this kind have been described by Payne, Fagge, Newton Pitt, and others. They tend to persist longer than the ordinary nodules

of children, and are decidedly more apt to be tender on pressure. Newton Pitt and Middleton have further described cases exhibiting extensive thickening of the affected parts, rather than discrete nodules, and this condition was attended in Pitt's case by a marked degree of peripheral asphyxia.—A. E. G.] The clavicle may be thickened and sensitive when the sterno-clavicular joint is enlarged.

Many neuralgic sympathies cluster around disease of the temporo-maxillary articulation. The nerve-supply to the joint comes principally from the infra-maxillary division of the fifth cranial nerve; this explains the occasional spasm in neighbouring muscles, with sundry reflex difficulties in mastication and even in deglutition. Another fine nerve thread goes to the joint from the auriculo-temporal branch of the infra-maxillary division; and hence, perhaps, the glancing pains about the temple and ear.

There is frequent deafness, probably from ankylosis of the ossicles of the middle ear. Iritis and conjunctivitis occur more often among people who are rheumatic than among those who are rheumatoid.

There is no visceral lesion which has not been described as an intercurrent disorder; none is caused by rheumatoid disease as such. Of the constitutional symptoms of the disease, we should try first to distinguish those which are part of the anterior bad health that originally brought on the disease; then those which follow anæmia, indigestion, and lack of muscular exercise; and, lastly, the special depression of spirits which generally ensues on the invasion of the system by an incurable disease. The heart should always be carefully examined; when it is affected the supposition of a rheumatic origin of the rheumatoid disease is strengthened. But in any arthritic disease of later life, structural and valvular deterioration of the heart may be but one more feature in the tendency to general decay. In private practice, as a rule, I have examined the urine of every rheumatoid patient, but without discovering any deviation from the normal which could be pathologically associated with the rheumatoid condition. But the specific gravity is usually low, and the excretion of urea under the normal mean; this may or may not be due to defective kidneys. The bowels are often obstinately constipated.

This sketch of the clinical history of rheumatoid arthritis has been drawn from well-marked cases. But the transitional forms are not unimportant: sometimes indeed the landmarks are so confused that it is almost impossible to make out where we are. A discussion of a few such forms may be profitable.

It is not always easy to determine whether we have to do with an arthritis or an arthropathy. The latter term is more properly applied to arthritic phenomena which are non-inflammatory and apparently non-diathetic.

Some forms of arthritic disease which have all the essential elements of an arthropathy are obscured by intercurrent effusions. A quiet, dull arthropathy puts on a morbid activity for a little while, and speedily recedes again into quiescence. The local waves of pyrexia—very

short, perhaps, in duration—leave the joint sensitive and gradually more crippled.

In ingravescent cases there are periods of unexpected improvement. On occasion an arthropathic patient may do much more with the arms, or may walk better; but the ameliorative force is soon spent.

A monarticular lesion tends in certain cases to become symmetrical. An affected hip or shoulder rarely lacks some sympathetic tenderness or disability on the other side. Or a person between 70 and 80 years of age may be so young in habit of body that a lesion which begins as a monarticular disease of the shoulder creeps down to other and smaller joints, and finally invades more or less of the whole limb. The following is a case of an opposite kind. A maiden lady had rheumatic fever severely in childhood. The disease returned several times in a mild degree during the early years of adult life. Then it seemed dormant for a while. But a special stress on the nervous system and on the general health started a rheumatoid lesion in one hip-joint simulating a pure monarticular disease in an elderly person.

Some rheumatoid people, in whom a polyarticular disease is exceedingly mild, suffer disproportionate pain. This requires a special treatment, for life is made miserable by disturbance of sleep and of other functions.

In severe cases it often happens that the hands are not so much affected, and, although unable to walk or to use the arms, the patient may be able to knit or to write. Sometimes the disease stops in its course after involving only two or three joints; when a quiescent stage of this sort has been reached, with freedom from pain, comparative health may be enjoyed.

A patient who has no conspicuous atrophy may feel as if all the strength went out of her legs, a "kind of weak feeling down the front of the thighs, which causes her to have the sensation as if she cannot walk." It does not last long, and it usually occurs while walking.

The vagus storms of "gastric crises" or "bronchial asthma" may alternate with pain or tenderness in the joints; the one group of symptoms waxes as the other wanes, yet without apparent connection. The same remark applies now and then to the muscular dystrophy.

Cautions and safeguards.—Looking at rheumatoid arthritis as a neurosis, we see in it two separable elements—the substantial and the collateral. Some of the collateral elements, although not in themselves of any gravity, are so common as to form almost a part of the disease; others may become injurious or even destructive. All occupy a definite position which has often been misunderstood. They are signs which indicate the nature of the arthritis; but they are not necessarily present. A case of rheumatoid degeneration may run its whole course, even to absolute decrepitude, without a symptom apart from the arthritic and muscular dystrophy. There may be no pain, no pigment, no perspiration; and the heart may never be disturbed even in the smallest degree.

It has been argued that there is no causal connection between the

substantial and the collateral symptoms. There are many people, it is said, who are pigmented and have no arthritis; many who have neuralgia in the limbs and no arthritis; while a permanently quick pulse without arthritic trouble is common enough. Much evidence is forthcoming, however, to show that the collateral symptoms (first described by me in 1888) are, when present, a positive help in diagnosis. Take a common example. A case of arthritis of a doubtful nature is before us; the arthritic condition by itself does not tell its own story. But there are aids to a trustworthy opinion somewhere; what are they? Almost always we shall find one or more if we look for them with a trained eye. (α) Count the pulse; there may be a steady tachycardia of between 90 and 120 in the minute. (β) Or there may be a growing melasma on the face; perhaps numerous spots and little stains on the arms. (γ) Or the hands may be always cold and sweating. (δ) Or there are neuralgic twinges in the upper and lower limbs.

Now my plea is that if any one of these symptoms be present beyond question, and if it can be proved to have been synchronous with the arthritis in beginning and progress, the nature of that arthritis is demonstrated thereby. I do not pretend as yet to explain the connection of the facts but only to record them; their interpretation will come by and by. The phrase which best expresses our transitional knowledge is that the collateral phenomena above described when they exist are valuable clinical signs.

The common failure to recognise rheumatoid arthritis in its early and tractable stage is easily explained. In its beginnings it resembles other maladies; its early symptoms are possibly blurred by the anæmia, or the menorrhagia, or the leucorrhœa, which are its remote causes; to say nothing of the tuberculous disease which may exist in other members of the same family. But the pallor and the weakness challenge attention, and if there be a heritage of rheumatism, or if there have been an imperfect recovery from an attack of rheumatic fever, grave suspicion ought to be aroused. During weeks and months after such an attack the joints and muscles (especially in women under forty) should be closely watched, chiefly those of the arms and hands. Persistent neuralgia over the ball of the thumb and on the ulnar side of the wrist is a declaratory sign which seldom misleads. At least once a week the pulse should be carefully counted; and a gradual rise to 90 in the minute, with some rise in blood-pressure (valvular disease of the heart being absent), is very characteristic. And then if yellow smears appear on the face, with freckles and sepia stains on the arms and hands, none but the ignorant and the blind can err.

How is it, then, that the grievous error is so often committed of calling a rheumatoid young woman gouty, and treating her as such? She suffers from headache and from neuralgia in other parts; her family history is blotted with tuberculous consumption or premature senility; she exhibits the sure notes of pigmentation, cold hands, and rapid pulse. The tender spots over the carpal and tarsal bones, especially if there be

a little swelling also, are regarded as signs of gout. The sallow complexion and the yellow smears are supposed to mean biliousness and stomach troubles; but the conjunctiva is luminously pale, and the urine may be almost colourless. Why should the rheumatoid young woman just described be called gouty? What features of gout does she wear? What wrong thread of inquiry or induction suggested the thought that gout was nursed in her tissues? We are allured by the easy teaching of old humoral doctrines; the lessons of neurology are hidden and more difficult to learn.

When atrophy of the upper limbs is an early symptom of rheumatoid arthritis, it might be confounded with the beginning of progressive muscular atrophy. The essential point which distinguishes these diseases lies, I think, in the presence or absence of sensory phenomena. These were often very real and impressive in the rheumatoid lesion; startling by their intensity, and disturbing to the health by their duration. The neuralgia puts on many forms, and is often mixed with vaso-motor derangements which are themselves allied to pain.

In any family in which there is a tendency to tuberculous consumption, the existence of menorrhagia or leucorrhœa among a group of quickly-growing girls should be regarded gravely. One of the group may be consumptive, and another (without apparent cause) may fall into a rapid neural arthritis. The former event seems natural, but the latter is among the things least expected; pain and sweating and atrophy may be in advance of the arthritic symptoms, and perhaps the disease reaches an incurable stage while being trifled with as only an "obstinate rheumatism." Before the medical attendant can grasp the problem the storm has come and is gone; its havoc is ineffaceable, and a potential bread-winner is crippled for ever. The deep alliance between tubercle and rheumatoid degeneration is intensely practical, and meets us in many ways.

A study of the various modes in which rheumatoid arthritis may begin suggests the pitfalls into which an unwary practitioner may stumble. The patient who is awakened in the night by sharp and sudden pain is most likely cheered by the misty diagnosis that her malady is only "rheumatism," and the usual remedies are used with no effect. Sudden weakness in the right hand, especially after toil in playing the piano or in writing letters, is ascribed to muscle fatigue or perhaps to gout. "Muscular rheumatism" is the cheap and facile explanation of all rheumatoid attacks which begin with a dominant myalgia, or with neuritis in the large muscles and aponeurotic sheaths. The risk is great that pain and difficulty in locomotion may be attributed merely to strained ligaments, or to a like result of a petty accident. And in the case of the hip-joint the risk is very great indeed. During several months, or even a year, there is, perhaps, an obvious limp in the walk; and an intelligent curiosity may discover that one limb is distinctly shorter than the other. Collect all the evidence at hand for the purpose of a right judgment; it may be struma or an arthritis of a common sort; but do not pass it by and call it "only rheumatism."

The condition which has most elements of fallacy concerns the dorsal and lumbar vertebræ. If there be ankylosis of the articular processes, and osseous bridges bind together the contiguous bodies of the bones, there will not only be a great hindrance to movement, but a pain radiating from the back in all directions. Now if there has been a period of unwonted quiescence after an advancing arthritic mischief in other parts, the spinal trouble may come as a surprise and perhaps a bewilderment. Those spectral phantasms, hysteria and neurasthenia, will be trotted across the clinical stage. All the obscure accidents which may happen to stomach, liver or uterus will be reviewed; but our diagnosis will turn on the rigidity of the spine and on the arthritic nature of the case in past life.

In true rheumatoid arthritis no uric acid has ever been discovered in blood: there is no deposit of urates in the affected joints; and no succession of attacks of rheumatism causes ulceration of cartilages.

On the practical side of this matter there is much to be said of exceeding interest. A patient comes before us of a spare habit of body, a slightly forward attitude, and a halting gait. We note the ovoid face, the melasmic tinge around the eye, and the shining yellow smear on the forehead. We feel the cold moist hands; we glance at the nodular fingers; and our diagnosis is made. In the imaginary patient now before us the arthritis is assumed to be the central postulate. We ask ourselves, What sort of arthritis is it? The accompanying evidence is accumulative in force and truth. Are there any corroborative signs? If there are, and they can be proved to have begun with the arthritis, they announce beyond debate the nature of that arthritis.

On the other hand, we must not apply the name "rheumatoid" to other and even graver maladies, such as the arthropathy of tabes or of hemiplegic limbs. Nor must we forget the possibilities of malignant disease; or that a joint may be crippled by a dislocation imperfectly reduced, or by the results of an old fracture.

If asked for a definite prognosis, we should recollect that in a rheumatic or tuberculous family rheumatoid lesions may run an appallingly swift course, deforming and disabling without mercy.

J. KENT SPENDER.

Treatment of rheumatoid arthritis.—Rheumatoid arthritis, if untreated, is essentially a progressive disease which in the great majority of cases tends to spread from joint to joint, successively involving fresh structures without in any way relaxing its hold upon those previously attacked. It is true that as the disorganisation of the joint structures proceeds the pain, often very intense in the earlier stages, frequently diminishes; and the movements of the eburnated surfaces upon each other may entail comparatively little suffering: so that, if the osteophytic outgrowths are not so bulky as greatly to restrict movement, a fairly useful articulation may be left. The results of treatment vary greatly in different cases. In some instances no course of treatment that can be

devised appears to have any controlling power over the progress of the malady. This is most commonly the case in elderly people in whom the disease is little more than a senile change; but such instances are unfortunately not rare among younger patients suffering from the more acute varieties.

More often judicious treatment has the effect of bringing the disease to a standstill, without, however, bringing about any marked amelioration. Such a result cannot be regarded otherwise than as satisfactory when we bear in mind the obstinate and inveterate character of rheumatoid arthritis.

In a third and far less numerous set of cases, although the mischief done by the morbid process cannot be entirely undone, a conspicuous improvement, which sometimes attains to an almost complete restoration, is brought about. The swollen joints diminish in size; the bursal swellings, so often present in their neighbourhood, disappear; the effusion is absorbed; and with the cessation of pain a degree of mobility, almost equal to that which had been lost, is obtained. The fact that such a degree of improvement is sometimes met with renders it essential that in any given case no effort should be spared to bring about such a result.

In order that the treatment of rheumatoid arthritis should have any chance of success the following requirements must be complied with:—

1. The treatment should be commenced as soon as possible after the appearance of the earliest signs of the disease.

2. It must be such as to maintain and increase the patient's strength, and all measures which have an opposite tendency should be avoided.

3. To be effectual it must be steadily continued over a period of months; or even, with short intermissions, for a year or two.

The modes of treatment available may be grouped under the following heads:—(i.) Treatment by means of diet, hygiene, etc.; (ii.) Climatic treatment; (iii.) Treatment by baths; (iv.) Internal administration of drugs; (v.) External and local applications.

(i.) *Diet and Hygiene.*—The question of diet is a most important one in connection with the disease under consideration, for whilst a suitable diet lends material aid in coping with rheumatoid arthritis, needless and injudicious restrictions tend, on the other hand, to favour its progress and extension. This is a point on which it is impossible to lay too much stress, for the superficial resemblances between rheumatoid arthritis and gout have given rise to a widespread impression that a dietary suitable for a gouty patient is suitable for the rheumatoid patient also. Thus the amount of animal food allowed is restricted, or meat altogether forbidden. Alcoholic beverages are also cut off as far as possible, with the result that the patient's strength is still further impaired, and he or she is brought into a condition which favours the advance of the malady. Experience shows that an entirely opposite course is that which affords the best chance of arrest, and that no greater mistake can be made than to treat the sufferer from rheumatoid arthritis as if he were an ordinary gouty subject.

In the former disease the great need is to increase the patient's strength, or, at least, to maintain it as far as possible; and, accordingly, no restriction should be imposed other than this, that the food should be such as the patient can best digest and best assimilate. If dyspepsia be present it should be treated by drugs, and by such an arrangement of diet as is best adapted to the patient's digestive powers, whilst retaining as far as possible its nutrient properties.

Generally speaking, meat, far from being avoided, should be taken freely, together with a suitable quantity of vegetable food.

There are exceptions, of course, to the above general rule. When the temperature is continuously high, or when, as is more often the case, attacks of fever of no long duration occur, the diet must, of course, be modified accordingly, but should be as nutritious as is possible under the circumstances.

The affection of the temporo-maxillary joints, so commonly present, but usually no more than a transitory stiffness, constitutes in some exceptional cases one of the most conspicuous features of the disease, and one of the chief sources of anxiety to the medical attendant. If the teeth can only be separated a fraction of an inch the difficulty of feeding may be very great, and mastication impossible. Under such circumstances meat must be given in a minced or pounded form, and in small quantities at a time. In extreme cases liquids only can be taken, and the removal of teeth to allow of the introduction of liquid nourishment may have to be considered; or, again, the possibility of giving relief by surgical procedures. In such cases the limitation of movement, which, as specimens in our museums show, is in part, at least, due to osteophytic growths around the joints, is greatly increased by muscular spasm. Under an anæsthetic the jaws may usually be separated to an extent which is surprising to those who have had the patient under ordinary observation.

As a general rule a moderate daily supply of alcohol, unless contraindicated for any special reason, appears to be useful in cases of rheumatoid arthritis. Among alcoholic drinks the more generous wines are to be preferred to spirits; and when the patient is able to take it without discomfort a glass of stout at mid-day is often advantageous, especially to the younger classes of sufferers.

Atmospheric changes and exposure tend materially to increase the articular pains; and woollen underclothing adapted to the season of year should always be worn.

Exercise in moderation is to be recommended; and even when the joints of the legs are affected a certain amount of walking should be allowed; but this should not be sufficient to cause lasting pain. Even when the patient can only get about in a wheeled chair fresh air is desirable; for it must be our aim in this, as in other matters, to keep the patient in as good a state of general health as possible.

(ii.) *Climatic treatment.* — A warm, dry climate and a dry soil are the most suitable to rheumatoid patients, and the climate should be

as equable as possible, as sudden fluctuations are apt to increase their pain. If the patient's home is in a damp neighbourhood, removal to a drier locality should be urged; and well-to-do sufferers often obtain benefit from wintering in a warmer climate than our islands afford. In the choice of a place of sojourn, the importance of an equable warmth, such as can hardly be obtained on this side of the Mediterranean, should be borne in mind. Such a change is specially adapted to cases in which the disease is not sufficiently advanced to cripple the patient; for when this stage is reached the discomforts of a long journey may counterbalance the good to be expected from a change of climate. Those who remain in England usually find that an inland climate is more suited to their condition than that of the sea-side, for sea-air is often found to increase the pains.

(iii.) *Treatment by baths.*—Patients suffering from rheumatoid arthritis constitute a large proportion of the frequenters of spas; and there can be no doubt that the external application of mineral waters is often beneficial, especially as an adjunct to medicinal treatment.

In almost all cases in which circumstances permit, the patient, in whom the disease is not already so far advanced as to preclude hope of material advantage, should be advised to undergo a course of thermal treatment in addition to other measures; and it may be advisable that the course should be repeated annually for several years. On the other hand, this form of treatment may be overdone; it has appeared to me that a short course is often of more benefit than a long one, and that the good derived from a first visit is sometimes imperilled or annulled by too early a repetition of it.

The spas which enjoy a reputation for efficacy in such cases do not belong to any one class, but differ widely in the mineral constituents of their waters. It seems to me that the mode of application of the waters is of more importance in rheumatoid arthritis than their chemical properties; and the accessory factor of climate appears to have no slight influence in determining the amount of benefit obtained.

The combination of the hot douche with massage, which had its origin at Aix-les-Bains, appears to be decidedly more useful in this disease than frequently-repeated immersion baths. At Aix itself the patients often receive marked benefit; and those who are unable to travel so far can now avail themselves of the Aix treatment at various British watering-places, notably at Bath, where the arrangements are excellent, and where alone in our islands a supply of water raised by nature to a high temperature is available. Harrogate and Woodhall Spa may also be mentioned among the British spas at which sufferers from rheumatoid arthritis may be advised to seek relief.

Peat-baths, such as may be obtained at Strathpeffer and at many continental resorts, are sometimes of service in relieving pain; and if, as is not infrequently the case, a marked degree of anæmia is a feature in the case, resort to iron springs, such as those of Schwalbach, Spa, or St. Moritz, may be desirable.

Vapour baths almost always give marked relief for the moment, and, by reducing pain and stiffness, frequently improve the patient's powers of locomotion greatly; but frequent baths of this character are not to be recommended, for, having a weakening effect, they are apt to be followed by an aggravation of the disease. This does not apply to the local vapour baths of the Berthollet type, in which steam is applied only to the neighbourhood of the joints.

Electric baths are not open to the same objections as simple immersion baths; and in skilled hands they have yielded good results. Dr. Lewis Jones, who is able to speak with authority upon this point, has kindly furnished me with the following brief summary of his experience upon this point:—"The electrical treatment of rheumatoid arthritis has been carried out on two distinct lines, and in both ways with some success. The older mode was by direct application to the affected joints and their neighbourhood, using the continuous current, and so setting up vaso-motor changes to modify the nutrition of the joints. The more recent treatment consists in general electrification by means of the electric bath, in the hope of changing the general state of nutrition of the whole system, and so, indirectly, of arresting the morbid process. The former plan should be tried for cases in which the stress of the disease has fallen upon one or few joints, and the latter when the disease is more general.

"My own experience has been chiefly with the electric bath. Alternating currents from the induction coil, or from an alternating dynamo-supply, seem to be the most useful. In this way I have treated a large number of cases, and I consider that electricity affords more relief to the patients than any other form of treatment. When the disease is recent, and has not yet caused serious deformity, its progress can be for a time arrested by electric baths, and the pain and stiffness can be very much relieved. A long course of treatment is required, two or three months, for example, with baths twice or thrice a week. It is partly on this account that I have had better results in hospital practice where considerations of expense do not limit the attendance of the patients. My experience with early rheumatoid arthritis and the electric bath has been uniformly favourable as far as relief of pain and stiffness is concerned, but in inveterate cases of many years' standing it appears to be too late to hope for anything more than slight temporary relief, and I have not had any clear instances of the treatment having led to the complete disappearance of the arthritic tendency."

Dr. Jones adds that the baths should be resorted to as early as possible after the onset of the disease, and regards their postponement to the later stages as unfair both to the patient and to the method. [*Vide* article on "Electro-therapeutics," vol. i. p. 349.]

(iv.) *Treatment by drugs.*—The drugs which are our most potent weapons in the treatment of true rheumatism and gout have little or no efficacy in checking the development of rheumatoid arthritis. Sodium salicylate has in certain cases a marked influence in reducing severe articular pain, and on this account may often be given with advantage for short

periods; but it is unwise in these cases to give this drug continuously for a long time.

For the relief of the acute paroxysmal pain which results from muscular spasm I have found hyoscyamus of conspicuous value. The drugs which are chiefly relied upon as the mainstay in the treatment of rheumatoid arthritis are iodine and the iodides, and arsenic. Iodide of potassium is often prescribed, but its continued administration in such cases, for such long periods as are necessary, is not without its disadvantages; and the iodide of iron, either in the form of syrup or pill, is to be preferred. I have no hesitation in assigning to iodide of iron the first place among the medicines which I have employed in rheumatoid arthritis; but, like other drugs, it is useless unless it be perseveringly taken over a period of several months, or even for a year or more.

Arsenic may be given either alone or together with iodide of iron; and as in order to produce any effect it needs to be taken for a long period, the treatment should be interrupted for a short time at stated intervals. Cod-liver oil, either alone or in one of the many preparations in which it is combined with maltine, is a useful adjunct to other treatment; and when the patient is able to take it without discomfort, it should be continued at least throughout the colder seasons of the year. The effects of medicinal treatment, as of other measures, are most marked in the cases in which the disease appears in early life and in its more acute forms; but the patient should be instructed to persevere, even if at first there be no obvious improvement, or indeed the reverse; for it is a common experience that even in the cases in which the most satisfactory results are ultimately obtained no effect is apparent for several months after treatment is commenced.

(v.) *Local treatment.*—In rheumatoid arthritis local treatment is of little avail; even when it is applied for the relief of pain the results are usually disappointing, and it has no influence upon the course of the malady.

Continued dry massage, as distinguished from a short course of douche-massage, has the advantage of restoring the wasted muscles; but it is not of any material benefit to the affected joints, and if too long continued sometimes seems to do harm rather than good.

For the relief of pain anodyne liniments of belladonna, chloroform, and the like may be applied; or an old prescription, originating with Haygarth who first described the clinical features of the disease, may be tried, namely, covering the painful joints with hot sand.

The joints may be wrapped in cotton wool, or woollen knee-caps may give relief. Splints are to be avoided as far as possible; but when, as is seldom the case, they seem to be required, they should be constructed of cardboard, or of some other material which affords some support without altogether preventing the movement of the parts.

Finally, there are cases in which some morbid condition is present which, by its weakening effect, appears to offer a sufficient cause for the onset of rheumatoid arthritis; such as metrorrhagia, or other form of uterine disorder. When this is the case the attempt should first be

made to relieve the primary malady; for, while it is present, there is little chance that treatment directed against the articular disease, which appears to be its result, will be of any avail: indeed, the measures employed may even tend to increase the original evil.

ARCHIBALD E. GARROD.

REFERENCES

1. ADAMS, R. *On Rheumatic Gout*, 1857 and 1873.—2. BAKER, W. MORRANT. *St. Barthol. Hosp. Reports*, 1877, xiii. p. 245; 1885, xxi. p. 177.—3. BANNATYNE, WOHLMANN, and BLAXALL. *Lancet*, 1896, i. p. 1120.—4. CHARCOT, J. M. *Thèse de Paris*, 1853.—5. *Idem. Maladies des Vieillards*, 1868.—6. CHAUFFARD and RAMOND. *Revue de méd.* 1896, xvi. 345.—7. DUCKWORTH, Sir DYCE. Heath's *Dictionary of Practical Surgery*, 1886, i. p. 293.—8. *Idem. Brit. Med. Journal*, 1884, ii. p. 263.—9. FOX, R. F. "The Varieties of Rheumatoid Arthritis," *Lancet*, ii. 79, 1895.—10. FULLER, H. W. *On Rheumatism, Rheumatic Gout, and Sciatica*, 3rd ed. 1860.—11. GARROD, Sir A. B. *Gout and Rheumatic Gout*, 3rd ed. 1876.—12. GARROD, A. E. *Treatise on Rheumatism and Rheumatoid Arthritis*, 1890.—13. HAYGARTH. *On Nodosity of the Joints*, 1805.—14. HEBERDEN. *Commentaries*, 1804.—15. HUTCHINSON, JONATHAN. *Trans. Path. Soc.* xxxii. 1880-81, p. 193.—16. LANDRÉ BEAUVAIS. *Thèse de Paris*, 1800.—17. LANE, ARBUTHNOT. *Path. Soc. Trans.* 1886, xxxvii. p. 387.—18. MIDDLETON. *American Journal Med. Sci.* Oct. 1887.—19. ORD, W. M. *Trans. Clin. Soc.* 1879, xiii. p. 15.—20. *Idem. Brit. Med. Journal*, 1884, ii. p. 268.—21. PAYNE. *Brit. Med. Journal*, 1883, i. p. 622.—22. PITT, NEWTON. *Clin. Soc. Trans.* 1894, xxvii. p. 54.—23. SCHÜLLER, MAX. *Berliner klin. Wochenschr.* 1896, xxx. p. 865.—24. SENATOR. Ziemssen's *Handbuch*, 1875, 2nd ed. 1879, vol. xiii. Hälfte 1, article "Arthritis Deformans."—25. SPENDER, J. KENT. *Osteoarthritis*, 1889.—26. *Idem. Med. Soc. Proc.* 1888, xi. p. 209.—27. *Idem. Lancet*, 1889, ii. p. 947.—28. WICHMANN, R. *Der chronische Gelenk-Rheumatismus, etc.*, 1890, 2nd ed. 1892.

A. E. G.

RHEUMATOID ARTHRITIS IN CHILDREN

RHEUMATOID ARTHRITIS is a rare disease in children, although, since Moncorvo published the first monograph on the subject in 1879, a considerable number of cases of supposed rheumatoid arthritis in children have been recorded. I have myself seen nineteen cases. These occurred in children of ages ranging from two to thirteen years, and would all have been included by many observers under the one head of "Rheumatoid Arthritis." Further distinction, however, is necessary. It is probable that three quite distinct joint affections have been described under this name.

The least rare of these affections (A), the one most commonly described as rheumatoid arthritis in children, presents important differences both in its clinical symptoms and morbid anatomy from the disease seen in adults. Next in order of frequency is (B) a joint affection which, clinically at least, appears to be identical with the rheumatoid arthritis of adults. The rarest of the three affections (C) is one which is in reality quite distinct,

though it may closely simulate either of the preceding: it is an exceedingly chronic form of rheumatism proper.

A. The disease mentioned first may be defined as a chronic progressive enlargement of joints, associated with enlargement of lymphatic glands and spleen.

Its **causes** are unknown. Heredity seems to play no part in its causation. Bad feeding, privations, and faults of hygiene were by no means prominent in the cases observed.

The onset is almost always before the second dentition: ten out of twelve cases began before the age of six years, and eight of these began within the first three years of life; the earliest was at fifteen months.

Girls are somewhat more liable to be affected than boys: seven of the twelve cases were girls, five were boys.

Symptoms. — The onset is usually insidious, with stiffness of one or more joints, which slowly become enlarged; but occasionally the onset is acute with pyrexia, and it may be with rigors. The character of the joint enlargement is almost constant. It suggests rather thickening of the tissues round the joint than a bony enlargement. There is none of the bony irregularity of rheumatoid arthritis, and the absence of bony thickening and lipping, even after the disease is considerably advanced, is striking. Bony grating cannot be obtained, but creaking of tendon or cartilage is frequently present. Effusion is seldom a marked feature. Tenderness if present is usually slight. There is generally some limitation of movement. The child whose photograph is shown here was completely bedridden at the age of four years owing to an almost rigid flexion of all the larger

FIG. 4.—Chronic arthritis with enlargement of lymphatic glands and spleen, in a girl aged four years.

joints.[1] The joints earliest affected are the knees, wrists, and cervical spine. The fingers soon become affected, and later the toes. The sterno-clavicular and temporo-maxillary joints are but rarely implicated. The disease is probably never limited to one joint; it is almost always symmetrical. There is no tendency to suppuration in the joints, nor to bony ankylosis.

The muscles of the limbs waste considerably as the disease progresses: they may undergo contracture; thus the hamstrings became shortened in a case where the knee was kept constantly flexed. The electrical reactions are unaltered.

Enlargement of the lymphatic glands is a constant symptom. It affects primarily and chiefly those related to the affected joints, but may become more general. The glands are separate, rather hard than soft, not tender, and show no tendency to break down. They may become so large as to be visible, but more often do not become larger than a hazel-nut. Their size varies with the progress of the joint affection; improvement in the joints is followed by diminution in the size of the glands.

Enlargement of the spleen is almost always detected. It was found in nine out of twelve cases. The edge of the spleen is felt 1 to 1½ inch below the costal margin. The enlargement is roughly proportionate to that of the glands, and varies with the joint condition.

The heart shows no evidence of valvular disease, but hæmic bruits are sometimes present. A tendency to inflammation of pleura and pericardium seems to exist in these cases: in three cases this was shown post-mortem by old adhesions, and in two other cases there were physical signs suggestive of adherent pericardium. Moderate anæmia is generally present. Sweating is often profuse. A curious occasional symptom is slight prominence of the eyes; it is not associated with any enlargement of the thyroid gland. In cases beginning before the second dentition there is often a marked arrest of bodily development: a child of 12½ years of age in whom the disease began at four years of age still had the appearance of a child of six or seven.

The temperature in some cases is almost continuously raised to 100° or 101°; in others short periods of pyrexia alternate with longer periods of apyrexia.

The course of these cases is almost always slow. Temporary improvement is common; but, so far as I am aware, no recovery has been recorded: the child in the course of months or years becomes an almost helpless cripple.

The disease is not in itself fatal. Three deaths have occurred at the Hospital for Sick Children; two at the age of 3½ years, one at four years,

[1] I am indebted to Dr. Lees for kind permission to make use of this and other cases which were under his care at the Hospital for Sick Children. I am indebted also to the kindness of Dr. Barlow, Dr. Penrose, and Dr. Garrod, who have allowed me to use cases which were under their care. The substance of this article was first published by me as a thesis for the degree of M.D. of Cambridge in 1896.

due respectively to bronchitis, erysipelas, and pneumonia. A curious result of certain accidental complications was observed; in one case catarrhal jaundice, in another scarlet fever, in a third measles, were followed by definite improvement in the joint condition.

The **morbid anatomy** is revealed by three autopsies which were made at the Hospital for Sick Children. In two cases, which had lasted fourteen months, the joints, which were markedly enlarged, showed only increased vascularity of synovial membrane and ligaments, with slight increase of synovial fluid and thickening of the capsule, which, in one case, was very obvious. In a very advanced case of three years' standing there was, in addition to thickening and increased vascularity of the synovial membrane, some pitting of the cartilage at its margin, giving it in some parts a worm-eaten appearance; little processes of the thickened synovial membrane fitted accurately into these pits, and thus the changes in the cartilage seemed to be secondary to the changes in the synovial membrane; the rest of the cartilage was healthy. There were some fibrous adhesions in the joints in this case. The fibrillation of the cartilage, which is so characteristic of rheumatoid arthritis, even in its early stages, was absent in these cases: osteophytic change, and exposure and eburnation of bone, were also absent. The glands and spleen showed considerable enlargement, but on section appeared normal; save for a few small ecchymoses which were seen in the substance of the glands. The pathology of this disease is uncertain; but some of the symptoms are at least suggestive of a microbic origin.

Diagnosis in the early stage is often a matter of extreme difficulty. Where the onset is acute the disease is generally mistaken for acute rheumatism, until the persistence of the joint affection and the enlargement of the glands and spleen reveal its nature. Where the onset is insidious the early affection of the spine may suggest spinal caries; and I have more than once known the difficulty in walking which was due to the early affection of the knees to be attributed to rickets. The symmetry of the joint affection, the large number of joints involved, and the absence of any tendency to caseation, will generally suffice to exclude tubercular disease. The joint lesion of congenital syphilis may closely simulate this disease; but usually the history and the presence of other symptoms of the inherited infection will distinguish them. The diagnosis from the disease known as rheumatoid arthritis in adults, and from chronic fibrous rheumatism, is often very difficult in the early stage; the points of difference are mentioned below.

Treatment is unsatisfactory. Cold and damp are to be avoided, as they certainly increase the subjective symptoms. Temporary removal from town to country air usually does but little good, but some benefit may be obtained from residence in a warm dry climate. No special diet has been found useful; the most nutritious diet that the child can digest should be given. Confinement to bed is to be avoided as long as possible, and the child should be encouraged to use the joints unless there be much effusion. Hot air baths, daily or at longer intervals, do good.

Extension by weights may be necessary to prevent contracture. Iron, arsenic, and cod-liver oil are the most useful drugs; internal administration of guaiacol has been followed by slight improvement.

B. RHEUMATOID ARTHRITIS, that is, a disease undistinguishable from the rheumatoid arthritis of adults, occurs more rarely than the disease above described. In children I have seen six cases only. Its clinical features are practically the same as in adults, so that it is unnecessary to describe them in detail here.

Rheumatoid arthritis, unlike the disease above described, generally begins after the commencement of the second dentition; I have known it to occur, however, in a child twenty months old. Its causation is as obscure in children as in adults. Depressing conditions, such as poverty and insanitary conditions, were strikingly absent in some of the cases observed. In one case the disease seemed to follow acute rheumatism; but this is certainly not a common sequence. Four cases occurred in girls, two in boys. The joint affection is multiple: the monarticular affection of old age is hardly known in children; but a case was recorded by Mr. Sydney Jones, in which, after an injury, one hip was thought to be affected with the disease in a boy aged 6½ years. The malady is by no means confined to the smaller joints; in some cases, indeed, the larger joints are affected first.

The very slow form characterised by Heberden's nodes is exceedingly rare in children. A French writer (Diamantberger) has collected four cases. I have twice seen nodosities, closely resembling those described by Heberden, in children with congenital syphilis.

Rheumatoid arthritis is distinguished from the disease described above by the absence of enlargement of glands and spleen; by the presence of bony thickening and lipping about the joints, and in some cases by the presence of bony grating.

I believe that no post-mortem examination of a child affected with rheumatoid arthritis has been recorded; but presumably the morbid anatomy is the same as in adults, and differs as shown above (A) from that of the previously described disease.

Treatment may produce marked improvement, especially in the early stages. Where it has been possible to remove the child to a dry, warm, equable climate, such as that of Las Palmas, I have known the most striking improvement to follow. Good feeding, the internal administration of arsenic, and the use of the hot air bath, have given good results.

C. CHRONIC FIBROUS RHEUMATISM seems to be the best name for a third disease which has probably been indiscriminately included under the head Rheumatoid Arthritis in Children. Its etiology and pathology are probably identical with those of acute rheumatism. It is very rare in children. The cases first described by Jaccoud were in young adults, and they occurred after repeated attacks of acute rheumatism. I have known it begin, however, at the age of 3½ years with insidious onset and with no acute attack at any time.

The joints show firm thickening, very suggestive of extra-articular fibrous change; they are little, if at all, tender. There is no evidence of bony change. Deformities may be present, very like those of rheumatoid arthritis, owing to the invasion of the neighbouring tendon sheaths by the fibrous process.

The nature of the disease may be evident from the presence of heart disease or subcutaneous nodules, as in the case mentioned, or from other rheumatic manifestations. There is no enlargement of glands or spleen. So far as I am aware, no post-mortem examination of a child with this disease has been recorded; but the fibrous change in the capsule of the joint, extending into the tissues around and leaving the cartilage healthy, was shown by an autopsy recorded by Brigidi and Banti (quoted by Dr. A. E. Garrod in his article on "Chronic Rheumatism," p. 59). Treatment is disappointing. Salicylates are useless, but tonics, especially the syrup of the iodide of iron, do some good; massage also may be of service. The general treatment is the same as of the preceding diseases.

G. F. STILL.

REFERENCES

1. BARLOW. "Chronic Arthritis in a Child," *Clin. Soc. Trans.* 1889, xxii. 328. —2. CHEADLE. "Rheumatoid Arthritis," Keating's *Encycl. Diseases of Children*, 1889, i. 822.—3. DÉVIC. "Du rhumatisme noueux chez les enfants," *Prov. méd.* Lyon, 1888, iii. 405.—4. DIAMANTBERGER. *Du rhumatisme noueux chez les enfants.* Paris, 1891.—5. DONKIN. *Diseases of Children*, Lond. 1893, p. 216.—6. ELOY. "Du rhumatisme noueux infantile," *Un. méd.* Paris, 1883, xxxvi. 977.—7. FINLEY. "Chronic Polyarthritis in a Child," *Montreal Med. Journ.* 1896, xxv. 2.—8. GARROD, Sir A. B. *Gout and Rheumatic Gout*, Lond. 1876, 3rd ed. p. 502.—9. GARROD, A. E. "Arthritis deformans in Children," *Twentieth Cent. Pract.* Lond. 1896, ii. 551.—10. HENOCH. *Vorlesungen über Kinderkrankheiten*, 2nd ed. 1883, p. 728.—11. HUTCHINSON. "Osteoarthritis in Early Life," *Archives of Surgery*, 1894, v. 82.—12. JONES. "Osteoarthritis in a Boy," *Clin. Soc. Trans.* 1887, xx. 265.—13. KOPLIK. "Arthritis Deformans in a Child," *Archives of Pædiatrics*, 1896, xiii. 3.—14. LACAZE-DORÉ. *Étude clinique sur le rhumatisme noueux chez les enfants.* Le Mans, 1882.—15. MATTHIEU. *Contribution à l'étude clinique du rhumatisme noueux chez les sujets jeunes.* Paris, 1884.—16. MONCORVO. *Estudo sombre o rheumatismo chronico nodoso na infancia.* Rio de Janeiro, 1879.—17. PELISSIÉ. *Le rhumatisme articulaire chronique progressif chez l'enfant.* Paris, 1889.—18. SCHNITT. "Bidrag till kannedomen om arthritis deformans hos barn," *Finska läk.-sällsk. handl.* Helsingfors, 1888, xxx. 459.—19. STOICESCO. "Le Rhumatisme noueux chez l'enfant," *Progrès méd.* Paris, 1876, iv. 287.—20. WAGNER. "Ueber Rheumatismus chronicus und Arthritis deformans im Kindesalter," *Munch. med. Wochenschr.* 1888, xxxiv. 195.—21. WEIL. *Nouvelle Iconographie de la Salpêtrière*, 1890, i. 16.

G. F. S.

RICKETS

SYNONYMS—*Rhachitis, Rhachitismus, Morbus Anglicus* (*Englische Krankheit*).

Short description.—Rickets is a general disease of early childhood affecting the nutrition and development of the whole of the growing organism. The most marked physical changes are seen in the bones. Ossification, which is still in active process, is both retarded and perverted. The bones grow irregularly, and, remaining largely cartilaginous and soft, yield under traction and pressure, thus giving rise to various distortions and deformities. The evolution of the teeth is delayed, their structure is fragile, imperfect, and subject to early decay. This striking affection of the osseous skeleton has concentrated attention upon one special feature of the disease, and led to too narrow a view of its pathology. Rickets has until recently been regarded as chiefly a disease of bones; it has been thus classed in the text-books, and its etiology and pathology considered almost entirely in this relation. The defect of growth and nutrition is, however, by no means limited to the bony framework of the body: all the chief structures are involved. The muscles are wasted and remarkably enfeebled, the ligaments relaxed. The mucous membranes exhibit an abnormal tendency to catarrh. The brain is functionally backward, while the reflex nervous system shows increased irritability, so that various forms of spasm and convulsion are readily excited. The blood is impoverished, its red corpuscles being diminished to such a degree that in some cases the anæmia is well marked. Lastly, in some cases there is fibroid enlargement of the liver and spleen.

History.—The name, primarily derived from the Old English verb *wrikken*, to wrest or twist awry, arose no doubt from the application of a term commonly applied in the country to inanimate structures, to a disease whose most striking characters are contortion and deformity. The ill-formed child with the soft and loose-jointed framework of its body bent and giving way was called rickety, just as a dilapidated table or chair was so styled, and the condition received the name of "The Rickets." Glisson, who first described the affection in the seventeenth century, proposed the conversion of the common term "Rickets" into "Rhachitis," partly on account of the similarity of the sound, partly as a derivation from the Greek ῥάχις, a spine, on the ground that the dorsal spine is one of the first parts attacked. This, rather than Trousseau's derivation from the Norman word *riquets*, appears to be the correct origin of the English name.

Rickets was recognised on the Continent soon after Glisson's description of it in England, whence it was supposed to have spread; but it has no doubt existed in Europe from early times as an accompaniment of

civilisation, and it is now becoming common in the younger countries of America and Australia.

Symptoms.—The appearance of a child suffering from well-marked rickets in its ordinary form is distinctive. The square forehead with rounded, boss-like projections on the frontal bone; the enlarged ends of the long bones—the deformities produced by the giving way of soft and yielding bone, such as the contracted chest, the deformed ribs, the curved spine, the protuberant abdomen, the bent arms and clavicles, the bowed legs or knock-knees, the yielding ankles, the defective, decaying teeth, the stunted growth—are features which cannot be mistaken. In many

FIG. 5.—Rickets.

C. G., æt. 2 years 6 months, showing general deformity and distortion of the bony skeleton, muscular wasting and debility. (Hospital for Sick Children, Great Ormond Street.) Photographed from life.

instances the child is plump or even fatter than normal, others again are thin, wasted and puny.

In addition to these peculiarities of outward form, the rickety infant is pallid, flabby, and feeble. Muscular debility is indeed one of the most constant and characteristic features of the disease, although little stress has been laid upon it. In severe cases the child, at two years of age or later, is unable to stand or walk, or even to sit up; if it has been able to walk it has lost the power. Such cases are not infrequently mistaken for paraplegia. Sir W. Jenner relates an instance in which the patient, a girl of six, could not change her position in bed or lift her arm an inch without assistance.

The feebleness of muscle and softness of bone interfere with the action

of respiration, and the lower portion of the chest falls in laterally with each contraction of the diaphragm; the feeble intercostals are unable to lift the soft ribs, which give way under the pull of the phrenic contraction aided by the external atmospheric pressure. This inefficiency of the respiratory mechanism greatly increases the danger of the pulmonary diseases to which rickety subjects are extremely prone. The affection of the bones in severe cases is sometimes accompanied by tenderness so marked that the child cries when handled, a sign possibly of the commencing scurvy with which rickets is occasionally associated. Another prominent symptom of rickets is profuse sweating, especially of the head, and during sleep; this is evidently accompanied by a sense of heat, for the child throws off its bed-clothes at night and lies uncovered regardless

FIG. 6.—Rickets.

From the same subject, showing contraction of thorax and prominence of abdomen. (Hospital for Sick Children, Great Ormond Street.) Photographed from life.

of the cold. There is, however, not only no pyrexia, but the temperature is, as a rule, subnormal; unless there be concurrent scurvy or some inflammatory complication, such as broncho-pneumonia. The abdomen is prominent owing partly to the depression of the viscera, partly to the gaseous distension of the intestines and the feeble condition of the abdominal wall. Moreover, the bowels are liable to be relaxed, the stools being loose and offensive; sometimes, in the absence of biliary colouring matter, almost white; sometimes green, sometimes of darker colour, and slimy. There is a tendency to bronchial and laryngeal catarrh, so that bronchitis and laryngitis are frequent complications. Nervous symptoms form a marked feature of the disease. Sir William Jenner first noted the exceptional tendency to convulsion, and the close dependence of the convulsions of infancy upon rickets was subsequently established by Dr. Gee. Reflex excitability is exaggerated, and probably the cerebral control of the imperfectly developed higher centres is also defective, so that

various forms of spasm are readily set up by slight causes of irritation. Laryngismus stridulus, tetany, and general convulsions stand in special relation to the rickety state.

Laryngismus stridulus is rarely met with apart from rickets, of which, although not universally present, it is a common symptom. In this condition the reflex apparatus of the glottis is so hypersensitive that spasm is excited by slight stimuli, not only by crying or laughter, but even by a breath of cold air, sudden movement, or the emotional disturbance produced by fright, anger, or the vexation of crossed purpose, such as the refusal or removal of a toy. Attacks are especially apt to occur on the child first waking from sleep, particularly in the early morning. The laryngeal spasm is marked by a sudden arrest of respiration, which lasts usually only a few seconds, and as suddenly ends with a prolonged crowing inspiration as air is again drawn through the narrowed glottis. The spasm varies in intensity; sometimes there is merely a slight crowing sound with each inspiratory effort for a brief space; or the muscles of respiration may remain fixed until the child is gravely cyanosed; occasionally death from actual asphyxia occurs before the spasm is relaxed. An attack of spasm of the glottis is not infrequently the precursor of a general convulsion, and must always be regarded as a significant indication that the condition of the nervous system has become unstable, and is one in which convulsions would readily be set up.

Tetany is a curious state of painful muscular contraction, a tonic spasm, chiefly of the hands and feet; it is also closely and especially associated with rickets. In these cases laryngismus is a constant accompaniment, and tetany often follows an attack of diarrhœa, to which rickety subjects are unusually prone.

It is true that tetany occasionally arises in adult life as a sequel of exhausting disease, as in women during lactation, and as a sequel to diarrhœa or typhoid fever. Such cases are, however, comparatively rare, and tetany is especially an affection of early childhood and of the rickety state. In tetany the thumbs are first affected, being adducted and drawn into the hollow of the palm, with tips pressing forcibly against the middle phalanx of the third finger, and even driven into the skin. The fingers are drawn together and overlap, and the palm is hollowed, so that the hand becomes cone-like, "the accoucheur's" hand of Trousseau. The wrist is slightly flexed, and in severe cases, when the muscular spasm is great, the back of the hand and wrist may become purplish, swollen, and even slightly œdematous, from the pressure of the contracting muscles on the venous circulation.

The feet are affected in like manner, but usually in less degree. The toes are adducted, flexed, and overlapping, and the forepart of the sole of the foot is hollowed and concave by the drawing inwards of its borders; the dorsum is arched, and in severe cases swollen, congested, and shiny from pressure, like the back of the hands.

In some cases the spasm is said to extend to the muscles of the trunk, causing slight opisthotonus, and occasionally to those of the jaws, causing

trismus; if this does ever occur, it is rare, and beyond my experience. The muscular cramps are painful, and when extreme acutely so; this we learn from the statement of adults, and it is noted, accordingly, that a child who suffers from severe tetany cries incessantly, and screams when handled.

The tetanoid state persists during sleep, and even under chloroform. It continues a considerable time, often for weeks, and is apt to recur. Remissions occur from time to time. These, however, are not complete; some degree of rigidity still remains.

The spasm can be excited afresh by pressure on the main artery or nerve—probably on the latter—for the motor nerves are in a state of hyperexcitability, not in the limbs only, but in other parts also. Thus, as first pointed out by Dr. Barlow, the sharp drawing of the forefinger over the skin in front of the ear, where the facial nerve emerges, in a child suffering from tetany is followed by contraction of the facial muscles. Percussion of a muscle causes a like contraction in it. The electrical excitability of the nerves of the most affected parts is also increased both to faradism and galvanism; and, as Erb has shown, the mode of reaction to the voltaic current is reversed, contraction being first excited by positive instead of by negative closure, and a prolonged "tetanus" contraction following both anodal and cathodal closure—the only condition in which anodal "tetanus" has been observed in man. It is said this increased neuro-muscular irritability may sometimes be found in rickety children who are free from attacks of tonic spasm.

Such are the symptoms and physical characters of rickets in its full and complete expression. All these features are not, of course, present in every instance. The disease is met with in every degree, from the smallest indications to the extreme forms.

Slight cases of rickets are liable to be overlooked. A little bending of the ribs, a prominence of the frontal bone, small areas of thinned elastic bone on the occipital or parietal, or soft edges of these flat bones, may be the only manifestations of rickets as far as the bony skeleton is concerned. Indeed, the rib-beads constituting the earliest signs met with in the fœtal state may be found alone in the early stage. The concurrence of head sweats, or laryngismus, or convulsion, or a tendency to catarrh, will be sufficient to establish the existence of rhachitis. In a more pronounced case there may further be some enlargement of the wrists, the girdle contraction of the chest, the bending of the long bones of the limbs. In the most complete and severe form the distortion of the skeleton may be extreme, and the whole series of other symptoms affecting the muscles, mucous membranes, and nervous system which have been described may perhaps be present; but the concurrence of all in the same subject is extremely rare.

Morbid anatomy.—The bone changes have been aptly described by Sir W. Jenner as exhibiting extensive preparation for ossification and imperfect performance of the process. My own observations agree in the main with the opinion that, as shown by Kassowitz, the rickety

process is not merely a defective ossification, but is accompanied also by a hyperplasia.

The Cranium.—The skull well exhibits the three great features of perverted ossification; namely, delay, atrophy and hyperplasia. The margins of the bones remain membranous; spots of thinning, the "craniotabes" of Elsasser, are perceptible in the parietal and occipital bones; and small bosses or swellings form symmetrically on the frontal, on the parietal and occipital, and even on the temporal bone. The atrophic lesions of craniotabes consist of wasting and thinning of the inner table, so that the inner surface of the bone, when the dura mater is removed,

FIG. 7.—Rickets. A case of extreme bossing of the skull, the rickety changes probably aggravated by congenital syphilis. Front view. Drawn from life.

A. W., aged 3 years and 6 months. (Hospital for Sick Children, Great Ormond Street, 1881. Dr. Cheadle.)

presents shallow depressions; some of these, in extreme cases, extend through to the pericranium, while the bone around is thin and elastic. These thinned areas have been variously attributed to pressure of the enlarged brain, and to that of the pillow; but they are probably patches of bone which have grown imperfectly. The elevations or bosses are developed chiefly from the outer table of the skull, and consist of red, highly vascular, spongy material which yields to pressure. These bony growths, in some instances, become more or less completely absorbed; but in most they become organised, and remain as rounded projections or bosses which are characteristic, or they may spread diffusely into more general thickening of the bone; in either case they largely assist in giving to the rickety skull its special character.

The chief features of this skull are as follows: the forehead is broad, square, and projecting, the eminences on each side being prominent and thickened; the top is flattened so that the head looks square or in some cases oblong, the parietal and occipital protuberances being well marked; a groove runs along the site of the suture between the two halves of the frontal bone, and is continued along the vertex to the anterior fontanelle, or beyond it, to the occiput. In some instances there is a distinct want of symmetry, as if the head had been twisted askew, so that the frontal region projects on one side and the occipital on the other. The head generally is larger than normal, the face by contrast looking smaller;

FIG. 8.—Rickets. A lateral view of the skull in the same case as the preceding. Drawn from life.

and the upper jaw is narrowed and elongated. The anterior fontanelle is large, and it remains open longer than usual; instead of being closed at eighteen or twenty months, it may be still more or less patent at two or three years of age; a slight opening may indeed be detected as late as five years. The closure of the sutures, except that between the two portions of the frontal, is delayed also; and there is irregular thickening of the margins of the bones, notably in front of the anterior fontanelle: deep venous grooves, often mistaken for open sutures, are found in the temporal region and elsewhere. The teeth come late; in extreme cases none may have appeared at ten or eleven months; they often come through in irregular order; they are fragile and delicate, deficient in enamel, subject to early decay, and fall out before their time.

The Thorax.—In the first place, the shape of the chest is modified by the softness of the rib bones and their yielding to pressure. Of these

changes one of the most characteristic is a depression which runs transversely from the lower end of the sternum across the chest on each side to the posterior margin of the axilla, about the line of attachment of the diaphragm. When any obstruction to respiration occurs, and in aggravated cases under ordinary conditions of respiration, the chest is further drawn in along this line with each inspiration. This is a result of the yielding of the ribs in the direction of least support. Another depression runs obliquely down the front of the chest along the line of junction of the ribs with the cartilages; and at the bottom of this depression, or outside it, are seen the "beads" or enlargements of the growing ends of the rib bones.

The result of this giving way of the soft bones of the chest wall under atmospheric pressure, at the points where the ribs are least sup-

FIG. 9.—Rickets.

From the same subject, showing beading of the ribs, transverse groove, and lateral depression of thorax. (Hospital for Sick Children, Great Ormond Street.) Photographed from life.

ported by the rest of the bony framework and the solid viscera, is the projection of the sternum forwards. This projection is rounded, not acutely angular as in the true pigeon-breast. The latter deformity results from the respiratory difficulties of whooping-cough, of repeated bronchitis, or of post-nasal adenoid growths, and may arise independently of any rickety softness of bone. When these obstructions to respiration occur in rickety subjects the sternal protrusion becomes more extreme.

The enlargement of the ends of the ribs at the junction with the costal cartilages—the "beads" which collectively form what is called the "rosary"—is the earliest of all the bone changes; the beads have been recognised at birth, and in some instances even in the fœtal skeleton. The beads are most marked in the lower ribs, and examination after death shows that they are more prominent on the inner than on the outer aspect of the bone. A slight degree of beading, however, may not be

abnormal. Posterior nodosities which appear to arise from partial fractures are also found near the angles of the ribs in severe cases. In addition to these changes in the thorax proper the clavicles are often thickened and more curved than normal; in some instances they present "green-stick fractures" or imperfect splintering with consequent thickening of the shaft.

The scapula in severe cases is curved, so that the posterior aspect is convex in conformity with the convexity of the back.

The Spine.—The back is rounded, owing to the relaxation of ligaments and the inability of the enfeebled muscles to keep it erect; sometimes there is a slight lateral curvature, and the forward lumbar curve is increased. In the early stage both these curves can be made to disappear by traction; but when the child begins to sit up, permanent deformity may be established if the rickety condition persist unrelieved.

The Pelvis.—The flat bones are thickened irregularly as elsewhere: when the body is supported erect on the limbs the pelvic arch, compressed between the weight of the body acting downwards from above through the spine and the upward resistance of the thigh bones, gives way; thus as a general result the pelvis becomes narrowed by the pushing forward of the sacral portion towards the pubes, and is more shallow.

The Long Bones of the limbs: Bones of the upper extremities.—The lower ends of the radius and ulna are thickened, both the epiphysis itself and the junction of this with the shaft; thus is constituted the enlargement of the wrists, which is one of the earliest signs of rickets.

Similar but less pronounced changes may be found at the upper end of these bones and at the upper and lower end of the humerus, being more marked at the latter. The changes observable are at first confined to these; but later, as pressure comes into play, and the child, in squatting or crawling, begins to lean its weight upon its hands, the shafts of the bones of the upper and forearm become curved and twisted. The force of gravity appears to be aided and modified by the support and traction of the muscular attachments. The "green-stick" fracture previously spoken of is not uncommon in these bones, and in rare instances complete fractures have been met with. In severe cases thickening of the ends of the metatarsal bones and phalanges has occasionally been observed.

The Bones of the lower limbs.—In these the earliest and most characteristic change, often indeed the only one to be found in minor examples of the disease, is enlargement of the lower end of the tibia from thickening of the epiphysis and its junction with the shaft. In the more pronounced cases the upper end of the same bone, and both extremities of the fibula and of the femur, are similarly affected in varying degrees.

When the child begins to crawl or walk about the results of weight pressure begin to appear, as in the case of the arms; and the long bones become bowed. At first the tibia alone grows a little concave on its

inward aspect; this increases if the child is allowed to go on walking and standing, and the femur becomes bowed in like manner. In some of the more extreme cases the tibia undergoes a forward curve just above the ankle, and the femur is arched forward in similar fashion. This is probably the result of pressure on the femur upwards and on the feet backwards as the child is carried in the arms.

Such are the general features of the bone affection and deformities in rickets.

Some of these abnormal conditions disappear with time and growth. In adult life the beads on the ribs are no longer recognisable, the enlargements

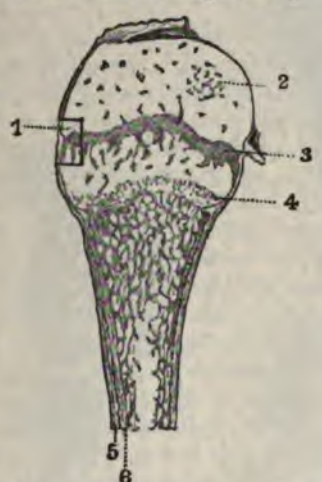


FIG. 10.—Vertical section through the upper half of the humerus of a child aged one year. Natural size. The rectangular area at 1 corresponds to the section, part of which, drawn under the microscope, is shown in Fig. 11. The cartilage of the upper epiphysis contains enlarged blood spaces, and at 2 a number of these are closely packed together, showing the commencement of a centre of ossification. The dark band at 3 represents the epiphyseal cartilage much broader than normal, and provided with irregular processes on its under surface. At 4 is the advancing plane of ossification; this also is broader than in the normal state, in which the layers 3 and 4 are not, as here, separated by a deep layer of soft tissue, but are closely interlocked, and together constitute a plane of tissue of no more than $\frac{1}{16}$th of an inch in depth. The number 5 indicates the periosteum and soft porous bone formed from it after the commencement of the rickets. The number 6 points to the periosteal bone formed before the commencement of the disease. (From an original drawing by Mr. Jackson Clarke.)

of the ends of the long bones undergo more or less complete involution, and the deformity of the chest tends gradually to become corrected; but an antero-lateral depression below the nipples persists to adult life in some cases.

The curvatures of the spine and of the long bones slowly straighten, and, if moderate, disappear altogether; although in more pronounced cases they remain during life. The contraction of the pelvis, however, when extreme, remains; and it forms not infrequently a serious source of difficulty and danger in parturient women. The bossing of the skull is toned down, although when the hyperostoses are large they remain in a modified form, and are prominent during life; the square, projecting fore-

FIG. 11.—Part of the other half of the same bone as that shown in Fig. 10, magnified with a low power. (Hartnack, oc. 4, obj. 3.) 1, Fibrous layer of the periosteum; 2, cellular layer of periosteum; 3, bone (imperfectly calcified) formed from the periosteum; 4, cartilage of the upper epiphysis, the disposition of the cells shows that they have multiplied equally in all directions; 5, upper limit of the epiphyseal groups of cells multiplying in a vertical direction; 6, a group of capillary blood-vessels surrounded by calcified cartilage matrix (the matrix of the cartilage is not indicated except where it is calcified); 7, a region similar to that shown in Fig. 10, but with multiplication of cartilage-cells in the calcified tissue which surrounds the blood-vessels; 8, points to the extremity of a blood-vessel where the periosteum dips inwards between the epiphyseal cartilage and the head of the bone; 9, represents part of the epiphyseal cartilage, which constitutes a deep layer in which the cells are irregularly arranged instead of the narrow plane of scalariform groups of cells seen in the normal state. (From an original drawing by Mr. Jackson Clarke.)

head and thickened margins of the sutures also continue as permanent indications of the extinct fault of structural growth which gave rise to them. When the rickety condition is severe, growth is defective and the stature short.

Histological changes.—The morbid changes in the more intimate structure of the bones consist essentially of modifications of the normal process of ossification; in excessive formation of cartilage and of the proliferating layer of the periosteum, in retardation of the development of bony tissue in these, and in perversion of the process. Bone is formed irregularly instead of by orderly advance of the ossifying column, and it is imperfectly calcified.

With this perversion of the formative process there goes also an absorption of bone already formed. In the long bones these abnormal conditions are exemplified by the greatly increased vascularity of the cartilage—vessels invade the cartilage from the periosteum, and in severe cases enlarge so greatly that they look like hæmorrhages or blood-spaces; and also by the excessive development of the proliferating zone of active growing cartilage cells at the epiphyseal end. The cells are increased in number and crowded together; sometimes they actually replace the matrix. There is also abnormal shallowness of the columnar zone, where the cartilage cells are being arranged into columns preparatory to their evolution into the medullary spaces of the forming bone. Further, these spaces are themselves irregular in level; and some advance obliquely in the shaft, thus impinging upon and destroying adjacent columns. In extreme cases this latter zone may be quite wanting, and spongy bone approaches the columnar zone of cartilage.

The bone formation takes place not only round the spaces but above and below them also, and small areas of bone may even be found in the unchanged cartilage. The medullary portion of the shaft is more vascular than normal, the interspaces larger.

The formed bone itself undergoes changes; the vessels enlarge and new ones form, which press upon the septa between the medullary spaces and cause their partial absorption, so that the spaces open into each other. Thus the strength of the bone is impaired, and the slight fractures occur which I have already described. As the septa of the formed bone are eaten away, fresh bone is formed which is deficient in lime. This deficiency is due, not to removal of that material as at first supposed, but, according to the later observations of Kassowitz, to defective deposit; the result is that rickety bone contains only 32 to 52 per cent of lime as compared with the 63 to 65 per cent in normal bone. As the active process subsides, vascularity of the growing portion lessens, and the spongy bone tissue becomes hardened and condensed by the further formation of new bone rich in lime salts. The junction of the epiphysis with the shaft is sometimes premature and the bone presumably shortened.

So far the description refers to intra-cartilaginous ossification; but changes from the normal occur also in the other form of bone formation, namely, in that from the periosteum.

The periosteum strips off the shaft of long bones more easily, and is more vascular than in health.

The outer fibrous layer is thickened, and the inner proliferating layer, in which the bone structure is developed, is the seat of active hyperplasia: the amount of the subperiosteal bone varies from the normal to as many as five or six layers, and it varies likewise in density and in the degree of calcification from spongy, limeless tissue to normal osseous structure. The periosteal formation, which is the sole form which obtains in the flat bones, shows excessive hyperplasia of the proliferating layer and imperfect ossification of like character. This hyperplasia is generally regarded as the result of an inflammatory process, a view which is supported by the fact that with the subsidence of the active rhachitic state more or less induration of bone—sclerosis—follows.

The Ligaments.—These suffer seriously in their nutrition; although the exact nature of the histological changes have not been investigated, they are obviously enfeebled, and, like the bones, yield under traction and pressure. Thus the ligamentous structures which bind together the bones of the feet give way as the rickety child begins to bear its weight upon them, and flat foot results. In like manner the ankles relax under the pressure and the feet splay out sideways; the knee ligaments give way, and knock-knee results; the supporting ligaments of the vertebral column yield, it becomes convex or curves laterally; and thus throughout all joints this laxness and feebleness of the binding ligaments and tendons prevail and alter shape and symmetry.

Muscles.—The muscles are also profoundly affected. This is shown by their feebleness, which, as has been stated, is in some instances so great that the child may be unable to stand or walk, keep the trunk erect, or in extreme cases to move in bed, or even to raise the head. If the structure of the muscles be examined they are seen to be flabby and wasted, and under the microscope the striation is blurred and indistinct. A complete microscopical and chemical investigation of the tissue changes is, however, still wanting to the morbid anatomy of rickets.

The Skin.—The skin may exhibit nothing abnormal except some slight pallor; but in severe cases the anæmia is well marked, and in these instances there is also pigmentation. The subcutaneous fat is frequently increased; indeed in the majority of cases of mild rickets the child is as plump or more plump than in health. In a minority of cases, where there is some general failure of health or a concomitant cachexia such as congenital syphilis, the fat is deficient, the child puny and emaciated. The existence of excessive sweating would seem to indicate the presence of some morbid product in the blood which acts as a stimulant on the glandular structures of the skin; or perhaps the morbid stimulation of gland function may act through the nervous supply, and be a consequence of that hyperexcitability of the reflex nervous system which is so conspicuous a feature of the disease, and is exemplified by the tendency to laryngismus, tetany and convulsions.

Mucous membranes.—These, throughout the whole respiratory tract and the whole extent of the alimentary tract, are especially prone to catarrh; so that laryngitis, bronchitis, gastritis, and enteritis are common complications. There is marked hyperplasia. The changes in the minute structure of the mucous lining, which are at the root of this abnormal tendency to catarrhal inflammation, have not been made out. All that can be said is that it is due to that vital instability and deficient resisting power which result from imperfect nutrition.

Lungs.—As a result of the falling in of the chest wall the lungs suffer mechanically. As previously shown, the depression is brought about partly by the pressure of the atmosphere and partly by the traction of the diaphragm upon the softened bony framework; and this is most effective where the thoracic wall is weakest, namely, at the junction of the ribs with the cartilages, and where the wall is least supported by the viscera. Tracts of collapsed lung are found beneath the line of the grooves of depression, especially under the beads; and with this a compensating emphysema is developed over the anterior borders, where the forward projection of the sternum removes support and promotes distension of the air vesicles. In addition to this collapse from direct pressure, there may be more extensive collapse from mucous obstruction when bronchitis has occurred. When cough is violent, as in whooping-cough, the collapse may be so great as to endanger life; and not infrequently, indeed, in that affection it is the complication which determines a fatal issue. The general catarrh of the mucous lining of the bronchi and respiratory tract in many cases has been already mentioned, but the exact changes of tissue which they betray have still to be determined.

The Heart.—In the case of the heart, also, whether in rickets any definite histological changes take place in the muscle has yet to be ascertained; but in view of the great feebleness of circulation which attends the rickety state there can be no doubt that this organ shares in the general malnutrition. The position of the heart in the chest is somewhat altered; by the falling in of the thoracic wall the apex is pushed a little outwards towards the left, where, as it comes into close contact with the bead in the rib, a white patch of thickened pericardium is formed by attrition. This is on the left ventricle, not on the right, as is the friction patch of the soldier.

The Liver.—In the majority of cases the liver is of normal size, although its margin may be low in the abdomen, owing to depression of the diaphragm by the contraction of the thorax. In certain instances, which, however, in my experience are comparatively rare exceptions, the organ is distinctly enlarged and harder than normal. This is due to a diffuse fibrosis, with slight increase of cellular elements and a deficiency in earthy salts, as in the bones. The hyperplasia is probably due to the chronic hyperæmia set up by obstructed circulation through the lungs, the result of contraction of the thorax combined with feeble heart power, collapse and emphysema, which retard the outflow from the portal system. Such hyperplasia is readily set up in childhood, a period when the

formative process is active. I have seen such fibrosis in marked degree in a child as the result of chronic bronchitis and emphysema. Or the hyperplasia may be irritative in origin, due to the circulation in the blood of some peccant material, such as has been held to be the active cause of hyperplasia in the bones and of the hyperactivity of the sweat-glands. In certain instances the hyperplasia is of syphilitic origin, especially when accompanied by great enlargement of the spleen.

The Spleen and Lymphatic Glands.—With the enlargement of the liver there is commonly also enlargement of the spleen, due to a corresponding fibrosis with increase of cell elements and decrease of earthy salts. As in the case of the liver, the hyperplasia depends either upon the mechanical hyperæmia due to obstructed pulmonary circulation and feeble cardiac power, to irritative stimulation, or to congenital syphilis. In cases where the enlargement is great, sometimes indeed so excessive that the lower border of the spleen extends below the umbilicus, it is almost certainly due to congenital syphilis. The appearance of such a spleen after death cannot be distinguished from that of the organ in so-called splenic cachexia, the origin of which is usually syphilitic, sometimes perhaps malarial in origin. The lymphatic glands in these cases are likewise increased in size, indurated, and fibrous from like interstitial and cellular change.

The Brain and Spinal Cord.—The increased size of the head in rickets is suggestive of a brain larger than normal; but this increase of size is due chiefly to thickening of the cranial bones. There is no evidence of increased skull capacity except in cases of separation of sutures when hydrocephalus is present. The actual condition of the brain, even as to size, is a matter of uncertainty; and of the histological changes we know little.

There can be no doubt that the nervous structures share in the general malnutrition; but whether this gives rise to increase of bulk, as in the growing ends of bones, or to diminution, as in the muscles, is not clear. According to some observers the brain is smaller than normal, and the extra space in the cranium is filled by effusion of fluid into the ventricles; according to others there is enlargement, due to increase of the neuroglia —a fibrosis such as that found in other organs.

The Blood.—The changes in the blood in rickets are imperfectly known. Anæmia is present, and as a rule it is proportionate to the other changes characteristic of the disease. When, however, the anæmia is extreme, it is associated with enlargement of the spleen, and is often out of all proportion to other changes; the red corpuscles are diminished in number, their shape and size irregular, and the hæmoglobin diminished. This extreme anæmia, coinciding with splenic enlargement, is probably the result of the special cachexia of congenital syphilis; although it must be allowed that such connection cannot be traced in all cases.

The Urine.—The analysis of the urine has yielded very diverse results. The great excess of lime salts stated to exist in it by early investigators

has not been found by the later, and it would appear that there is really no important difference between the urine of rickety and healthy children in this respect; nor can the presence of that lactic acid be detected which has been anticipated in accordance with a theory of the pathology of the disease which is no longer tenable.

Forms and Varieties of Rickets, and Conditions resembling it

Fœtal rickets.—Although, so far as I know, no microscopical record of progressive fœtal rickets exists, there seems to be no doubt, from the observations of Guérin, Tripier, Lauro, and others, that in certain rare cases rickets begins in intra-uterine life. The long bones are bent, fractures are found, and there is incipient beading on the ribs which on microscopic examination exhibit all the peculiar characteristics of rickety bone; the ossification of the cranial bones is also delayed. There are other cases in which bone changes, such as softening and partial fractures of the long bones, are found in the womb; but the microscope discloses no proliferating cartilage, no imperfectly calcified spongy bone, no true rickety aberration of structure. These cases have been traced to congenital syphilis.

Scurvy rickets. So-called acute rickets.—Rickets as a rule progresses slowly and insidiously; in a few cases it begins more abruptly, but the disease never sets in suddenly. In all cases in which this comparatively sudden onset has been alleged, there have been signs of bone changes and muscle weakness of longer standing which had attracted little notice until the process became more active.

There are, however, certain cases which have been called acute rickets from the rapid supervention of such acute symptoms as extreme tenderness of limbs, periosteal swellings, sponginess of gums, hæmorrhages. The periosteal swellings are due to subperiosteal hæmorrhages, and the condition has been shown by Dr. Barlow and by myself to be in reality one of scurvy, often, no doubt, superadded to rickets, but in its nature distinct. This affection, infantile scurvy, will be fully described in a separate article.

Syphilitic rickets.—When rickets arises in conjunction with congenital syphilis certain modifications follow. The child is as small, puny, and wasted as when rickets concurs with general starvation; and it will probably have the depressed nose and linear scars associated with congenital syphilis. Further distinctive features are the thinning of the flat bones, or craniotabes; and projections on the frontal and occipital bones, the so-called syphilitic bosses: these appear to be especially well marked in syphilitic cases, and, although met with in other instances in which no history or sign of syphilis exists, it is usually in minor degree. Dr. Barlow and Dr. Lees found a certain history of syphilis in 47 per cent; Dr. Baxter, in 75 per cent; in some cases there is hypertrophic fibrosis of liver and spleen and lymphatic glands.

Chondrodystrophia fœtalis. Achondroplasia. Fœtal cretinism.—Other cases, again, of so-called "fœtal" rickets, characterised chiefly by extreme and abnormal shortness of the long bones of the limbs, have likewise been found to lack the special features of rhachitic bone change. The area of proliferating cartilage is absent, and there is little or no columnar arrangement of cells, ossification taking place, in fact, almost entirely from periosteum; while the thickening of the ends of the ribs is not a cartilaginous over-growth, as in the true rickety head, but an osseous ring. From the peculiar fusing together of the bones forming the base of skull, the condition was regarded as one of fœtal cretinism; but later investigations, especially by Drs. Symington and A. Thomson, appear to render it probable that the condition is distinct both from rickets and cretinism, of which the chief features are arrested or defective enchondral ossification in intra-uterine life—chondrodystrophia fœtalis. The bones developed from membrane and those developed from cartilage late in intra-uterine life are normal; but those which are ossified from cartilage before birth are dwarfed. Thus, while the flat bones of the skull, formed from membrane, are normally grown—in contrast to the semi-membranous condition in rickets, with its hypertrophic bosses and atrophic craniotabes—the bones at the base of cartilaginous development are dwarfed and prematurely united. The ribs and pelvis are also defective, and all the long bones of the limbs shortened to half their usual length. The remainder of the cartilage-formed bones, which are developed chiefly after birth, are of normal size. In the majority of these cases the fœtus perishes in the womb or soon after birth; a few only survive.

Osteomalacia.—There is in true rickets a deposition of new bone which is deficient in lime, and when this process is extreme, as in certain cases where the general atrophy of other tissues is marked, the body is wasted and bones small; this condition has been called "rickety osteomalacia."

A case of the kind has, however, been described by Dr. Barlow, in which other marks of rickets such as rib beads, grooves, and cartilage proliferation were entirely wanting, and the morbid condition seems to be analogous to the osteomalacia of adults. [See art. "Osteomalacia," p. 147.]

Late rickets.—Rickets is a disease of the first two years of life, and in the majority of cases it makes its appearance before the end of the first year; in some instances, however, the disease does not set in until later. In one instance I had under my care a boy of ten, in whom the first manifestations were observed at the age of nine. The signs during life—namely, beading of the ribs, contraction of the chest, enlargement of the ends of the long bones of the limbs—were those of ordinary rickets; and post-mortem examination after death disclosed changes in the bones corresponding to those of genuine rickets. It is to be noted, however, as throwing some doubt upon its intrinsic nature, that the treatment by diet, so effective in this disease as it occurs in infancy, failed in this instance. Sir

W. Jenner has recorded instances of the first onset of the disease at a late period, namely, at the ages of three and nine.

FIG. 12.—Late rickets.

H. J. L., aged 11 years. (Case under Dr. Cheadle. Hospital for Sick Children, Great Ormond Street, 1880-81.) The disease commenced at 9 years old. The bones showed typical rickety changes. Post-mortem, *vide Pathological Soc. Trans.* 1881. Photographed from life.

Ætiology.—Rickets is the result of imperfect and perverted nutrition; so far pathologists are agreed: there is, however, some divergence of opinion as to the exact causes of this defect. Most of the hypotheses have

been based solely upon a consideration of the morbid changes met with in the bones, as if this comprised the whole pathology of the disease. To trace the nature and source of these faults of ossification may supply the key to the nature of the general affection, but no pathological doctrine can be regarded as satisfactory which does not also explain the morbid conditions of muscle and tendon, mucous membrane, and nervous system; these conditions are concurrent and constant, although they are less obvious and obtrusive than is the affection of the skeleton. The production of a rickety condition of bone by the application of Esmarch's bandage, as in the experiment of Kassowitz, does not produce the changes in the other tissues. The bones are certainly soft and they give way under pressure; but the ligaments are also soft and lax, and give way under pressure likewise; the muscles are atrophic and enfeebled; the mucous membranes catarrhal; the reflex nervous system hyperexcitable and unstable.

It is difficult to understand how the bone lesions and the other concurrent tissue changes can stand to each other in the relation of cause and effect, as has been suggested; or how morbid conditions, regularly associated with the bone lesion and in proportion to it, can be regarded as accidental. The explanation of the earlier and greater prominence of the bone changes lies in this, that the bones, being in the most active stage of their growth, exhibit the departure from the normal most clearly at a time when the coexisting changes in other organs and tissues are smaller and less visible.

Many are the causes to which the production of rickets has been attributed. Faults of diet, setting up gastro-intestinal catarrh, impaired digestion, vomiting and diarrhœa; want of light and fresh air; deficient clothing, dirt, and general bad hygiene; syphilis; inherited tendency, have all been credited with more or less reason as concerned in the genesis of the disease.

It appears certain that, as a rule, several factors are engaged, and amongst them many of those enumerated above. These factors, however, are not all in action in every instance, and they are not, therefore, all essential, nor equal in constancy and potency.

Climate, Season, and Locality.—Although rickets seems to exist in all parts of the world, its occurrence is clearly influenced by climate, and chiefly in respect of dryness, sunlight, and warmth. Thus it is most common in the temperate zone, especially in cold, damp, cloudy regions such as England, and certain portions of Germany, Italy, France, and North America.

It diminishes in frequency as high northern latitudes are approached, and, on the other hand, declines towards the south until it almost disappears in the tropics. In Australia, rickets was believed practically not to exist; but in 1891 it was shown by Mr. Muskett to be prevalent in the large towns, and cases have been met with even in the bush. The disease appears to be affected by season; cases are more frequent and more severe during the dark, cold winter months when the children live

largely indoors, and lack light, fresh air, and warmth; that is, when vitality is lowest. With regard to locality the chief point made out is, that the disease is, generally speaking, one of great cities—of London, Manchester, Liverpool, Glasgow, Vienna, New York; and that in this country it is especially prevalent in the towns of the great manufacturing districts of Lancashire and Yorkshire, the Black Country, Scotland and Wales; in these regions of smoke and darkness the mothers are largely workers engaged away from home, and their children are chiefly brought up by hand.

Influence of Sex.—Statistics as to the relative frequency of Rickets in boys and girls vary; but the general outcome of them seems to be that sex exerts no influence, that the disease is distributed evenly between the two sexes.

Inherited Tendency.—No satisfactory evidence has been produced to show that rickets is ever transmitted from the parents to the children. Rickets dies out with childhood, and is not likely to be handed down to the offspring of mature persons. The influence of heredity is probably limited to the transmission of a weakly constitution, or to some factor of imperfect nutrition of the fœtus in the womb. That heredity is not an essential or constant factor is shown by the fact that the children of perfectly healthy persons become rickety; in the vast majority of cases, indeed, rickety children are born of parents who do not exhibit the smallest trace of past rickets.

Congenital Syphilis.—It is now very generally agreed that rickets is not a simple expression of congenital syphilis, as M. Parrot contended; the evidence to the contrary is conclusive. In the majority of cases these children bear about them none of the well-established signs of congenital syphilis: the eruption, the snuffles, the linear scars, the pegged teeth, the keratitis are wanting. In many cases, moreover, the history seems absolutely beyond suspicion; and, conversely, many children who suffer from congenital syphilis are not rickety. It is clear, then, that syphilis is not a constant, invariable, essential factor. Congenital syphilis modifies rickets, it does not create it. The cases in which it does play a part have special features of their own. The child is puny and wasted, and it presents some of the distinctive signs of syphilis; and to these cases especially belong the craniotabes of Elsasser and the boss-like projections of the frontal and occipital bones in their most extreme form; although they are not absolutely limited to the syphilitic variety. Possibly the enlargement of lymphatic glands, liver, and spleen, met with in some cases, may prove to be rather a syphilitic than a rhachitic change.

Bad hygiene.—Defective hygienic conditions are largely concerned in the production of rickets. The great incidence of the disease upon the population of large cities and amongst the poorer classes there, and the fact that rickets is comparatively rare in the bright, sunny climates of the south, where life is spent largely out of doors in fresh air and sunshine, afford sufficient evidence of this. The want of sunlight and warmth appears to tell especially upon children of southern race when reared in

cold and uncongenial climates. The children of Neapolitan parents, for example, brought up in the great cities of America, are stated by Dr. Snow of Buffalo to suffer from rickets to such an extent that even those brought up at the breast do not escape. Foul air, want of light and sunshine, defective cleanliness, and lowered bodily warmth from scanty clothing favour the production of rickets by degrading nutrition.

Although, however, these influences of defective hygiene are frequently concerned in the production of rickets they are not constantly present, and are not therefore invariable or essential factors. Many cases of rickets arise in patients who live under excellent sanitary conditions, so far as air and light and cleanliness and warmth are concerned; a child may enjoy these in perfection and yet become rickety: and again a child may not become rickety, although brought up under the most unhealthy external conditions. These external conditions of defective hygiene must therefore be regarded as influential, but not essential; in extreme cases, however, they are generally at work.

Disorders of digestion appear to play a part in a number of cases; at any rate symptoms of gastro-enteric disorder—flatulence, vomiting, diarrhœa, and offensive stools—not infrequently precede the distinctive signs of rhachitic change. They are not, however, constant accompaniments; numbers of children become rickety who have no such antecedent gastro-intestinal disturbance, and numbers who do suffer from it do not become rickety. Further, when the disturbance is extreme, and vomiting and diarrhœa are severe and prolonged, the result is not rickets, but general atrophy; and examination after death in fatal cases shows little or no evidence of the characteristic changes in the bones.

It appears, then, that digestive disturbance of this kind is only effective when it is not extremely prolonged and excessive, and when it coincides with particular faults of diet. It probably acts by removal of certain special elements which are the least quickly and rapidly digested and absorbed. Digestive disturbance is not, therefore, an invariable and essential factor in the production of rickets.

Faults of diet.—The vast majority of cases of rickets arise in connection with errors of feeding. The fault of diet is not only the most common and potent cause, but sometimes it is the only cause. Rickets is produced as certainly by rhachitic diet as is scurvy by a scorbutic diet. This is seen in those by no means uncommon cases where children born healthy and of healthy, well-to-do parents, brought up under perfect hygienic conditions so far as air, light, cleanliness, and warmth are concerned, yet become rickety when brought up on artificial food. The only fault we can discover is the dietetic fault; and such cases are cured by a correction of the diet, without any other change of hygienic conditions; they are cured, in fact, by antirhachitic diet as certainly as scurvy is cured by antiscorbutic diet.

The effect of diet was shown in the most striking manner by Mr. Bland Sutton in the case of rickety animals at the Zoological Gardens; these animals got rapidly well on a change of one condition only, namely,

of food. Directly or indirectly, food is probably an invariable factor. The fault, moreover, is one of quality rather than of quantity. A child may be reduced by starvation to the last stage of atrophy, and yet not be rickety; and, conversely, it may be over-fed, fat and gross, and yet extremely rickety. There is a special fault of diet, one which produces a special defect of nutrition, and not necessarily general malnutrition. In this respect it is first to be noted that, in this country at any rate, rickets is practically unknown amongst sucklings. The only instance, within my own experience, of rickets arising in a child while at the breast during the first ten months of life was one in which the mother became pregnant during lactation; the suckled infant became rickety, the fœtus unborn escaped: rickets, then, may arise if the mother's milk be insufficient, or otherwise defective. Such cases are, however, undoubtedly rare. Even with congenital syphilis at work the child at the breast does not become rickety. If breast-fed children become rickety it is after weaning; the disease occurs almost entirely amongst children brought up by hand.

The exact nature of the diet fault which lies at the root of the rickety condition has been the subject of many hypotheses and much controversy. Certain broad facts, however, have been established with regard to it, which throw great light upon the matter. In the first place, children fed almost entirely upon farinaceous preparations—oatmeal, corn flour, bread, patent foods, with little or no milk, even if such diet produce no digestive disturbance—certainly became rickety. Similarly in the case of animals, Mr. Bland Sutton observed that the young monkeys at the Zoological Gardens in London, if deprived of their mother's milk and fed entirely upon vegetable food, chiefly fruits, become rickety. Two young bears fed exclusively upon rice, biscuits, and raw meat, of which latter they hardly ate, died of extreme rickets. It is not a diet limited to vegetable food only which is associated with rickets. The artificial production of rickets in young animals by Guérin, who substituted meat for mother's milk, although impugned by the later experiments of Tripier, has been remarkably confirmed by experience at the Zoological Gardens. For many years the lion whelps have been weaned early, and put upon a diet of raw flesh only; they have invariably become rickety to so extreme a degree that it has been found impossible to rear them. The condition is a true rhachitis; there is the same feebleness of muscle, the same debility, laxness of sinew and bending of bones; and the identity of the morbid changes has been fully established.

The potency of such diets in the production of rickets has led to many hypothetical explanations of the exact nature of the defect in them; whether this be negative or positive, the want of some necessary ingredient, or the presence of some noxious ingredient which perverts nutrition, especially that of growing bones.

The explanation which at first suggested itself was that as the bones are soft and deficient in mineral matter, and especially in lime salts, a want of lime salts in food is the cause of the deficiency in the bones.

Chossat and Milne Edwards produced curvature of the bones in animals by privation of earthy salts, but the characteristic features of rhachitic bone were found by Friedleben to be wanting in such cases. Others, however, as Voit and Baginsky, claim to have established the existence of true rickety change by such treatment.

While admitting that rickets may be produced artificially in animals by absolute privation of lime, yet that the want of lime, at any rate in the form of hydrate or carbonate, is not in itself the essential cause of rickets, as we see it in children, is proved by conclusive evidence. First there is the fact that rickets is extremely common in the limestone districts where the drinking-water is so heavily charged with lime that the children must necessarily take abundance of it; moreover, numbers of children become rickety who have lime water regularly added to their food. Secondly, there is the fact, according to Dr. Luff's analysis, that foods upon which children are especially liable to become rickety, such as the farinaceous foods for instance, are rich in lime and also in phosphoric acid; and cow's milk is richer in these than is human milk. So that not only will abundance of lime salts in the food not prevent the development of rickets, but as a matter of fact the disease is usually associated with a full supply of these materials.

The close association of rickets with a farinaceous diet suggested the idea that lactic acid might be the evil agent: starch, imperfectly digested, ferments and lactic acid is formed in excess, which, by uniting with the lime about to be deposited in the bones, is supposed to carry it off in soluble form: or, according to another view of its action put forward by Heitzman, it irritates the ossifying tissue and stimulates growth when the material necessary to complete the structure is wanting. Lactic acid is said to have been found in the tissues of rickety animals and in the urine; and Heitzman claims by its administration to have produced the condition directly. This formation of lactic acid, however, has not been confirmed; and the hypothesis is rendered improbable by the fact that rickets arises in children in whom there is no apparent disorder of digestion to favour lactic acid fermentation, who digest the starch or maltose thoroughly, and even wax unduly fat upon it. Moreover, according to repeated observations of my own, the rickety state disappears, and health is restored whilst the farinaceous diet is continued unchanged except by the addition to it of certain nutritive elements in which it is deficient. These clinical experiments show conclusively that the starch cannot of itself be actively harmful. Again, rickets arises in animals fed on a diet—such as lean meat alone—which is not productive of lactic acid; and, lastly, if lactic acid did exist in the blood it would be at once neutralised by alkali there. The fault in diet which is the chief factor in the production of rickets is clearly, then, neither deficiency of lime nor an excess of starch nor lactic acid generated from it.

An examination on the one hand of the foods on which children grow rickety, and on the other of the additions to diet by which the condition is cured, throws light upon this point. Certain defects appear to be

constant. An analysis of the foods on which rickets is most frequently and certainly produced—such as the various farinaceous foods, domestic or patent, with a small amount of milk, skim milk, condensed milk, artificial foods with desiccated milk, and the like—shows invariably deficiency in two of the chief elements so plentiful in the standard food of young animals, namely, animal fat and proteid. The only exceptions to this conclusion appear to be the cases in which the foods that do contain a sufficient quantity of these elements—as cows' milk, for example—produce digestive disturbance, vomiting, and diarrhœa, which disturbances lead to the loss of much of the material ingested. In such cases, moreover, the elements most slowly digested, those, that is, most slowly brought into a fit state for absorption—namely, the fat and caseine—would be most largely drained away. Not only so, but to substitute a food deficient in these more hardly digested fats and proteids, which, to ease the digestive difficulty, is almost always done, is to reach the same pathological result by a different route. At the Zoological Gardens the food on which young bears and monkeys become rickety—namely, biscuit, rice, and fruits—is markedly deficient in proteid, and fat is practically absent. The food of the lion cubs, which became rickety on a diet restricted to raw flesh, was almost destitute of fat, and was poor also in earthy salts, although rich in proteid. The meat was that of old horses almost entirely destitute of fat, and once a week lean goat's flesh. The bones were found to be proof even against the teeth of the adult lions, and those of the cubs were powerless against them, so that the cubs got from them neither marrow, fat, nor earthy phosphates. In this case the diet was not deficient in proteid, but in fat and earthy phosphates. The history of these lion cubs is very significant: with the exception of a single litter, suckled by the dam ten years before, the cubs brought up on horse-flesh in this way invariably died—the cause of death being, as invariably, extreme rickets. More than twenty litters had been lost in this way. The feeding of the last litter of lion cubs was begun in the usual fashion. The dam had very little milk, and at the end of two weeks the cubs were weaned entirely, and were then put on horse-flesh as usual. They quickly became rickety, and when I saw them at this juncture the muscular weakness, as well as bone deformity, were extreme. The malady advanced rapidly and one cub died. Then, by the advice of Mr. Bland Sutton, milk, pounded bones, and cod-liver oil were added to the raw meat, which was continued exactly as before; they were kept in the same dens with the same amount of warmth and light and air, and, with the single exception of the addition to the diet, no change of any kind was made in the regimen. The change in nutrition which followed was immediate and remarkable; in three months all signs of rickets had disappeared, and the animals grew up strong and healthy—a unique event in the history of the Society. The experiment seems a crucial one, and decisive as to the part played by fat and bone salts, with some caseine and lactine, in the production and cure of rickets.

That rickets frequently follows the prolonged food vomiting and diarrhœa provoked by cows' milk, is consistent with this estimate of the effect of deficiency of fat and proteid in its production. For as the fat must be emulsified or saponified before it can be absorbed, and as the proteid in like manner must be converted into peptone, these elements would be drained off rather than the lactine and salts, which are in solution and ready for immediate passage into the circulation. The result would be a great privation of fat and proteid.

Of the three elements of food the imperfect supply of which is found to be associated with rickets, fat is probably the most frequently, if not invariably deficient. The abundance of fat in milk, of the whole solids of which it forms one-fourth, points to the extreme importance of it in the nutrition of growing animals. If fat be removed from the milk, as in "skim" milk, rickets follows. The curative power of cod-liver oil is evidence in the same direction; and it is interesting to find in this connection, as recorded by M. Remy, that in Japan, where oils of fishes enter largely into food, and children are kept partly at the breast up to five years old, rickets appears to be unknown. Animal fat probably serves some special purpose in the nutrition of the growing structures of a young organism. It is found in all cells, it is probably essential to all cell life and growth, and it is further evident that fat formed in the body out of the carbohydrates cannot, for structural purposes, replace the animal fat supplied in food. Phosphate of lime, again, is essential to every tissue, and in rickets is found to be deficient in the bones and viscera. Proteid, again, is essential to the vitality of protoplasm, and indeed to the activity of all vital processes, and is therefore essential to the proper use of the other elements; but if it be in excess while the other elements are deficient, it may actually intensify the progress of rickets by stimulating the processes of tissue development which, in the want of other materials, cannot be duly carried out. Therefore a diet deficient in the element of animal fat and, in some cases also, deficient in proteid and earthy salts—one or both—would explain not only the faultiness of bone, but also the feebleness of muscle, the anæmia, the catarrhal tendency of the mucous membranes, and the nervous irritability. All tissues—not bone tissues alone—are ill-nourished if the above structural elements are deficient.

The general pathology of rickets may, perhaps, be summed up as follows:—Rickets is a disease in which all the leading structures of the body suffer both from defective and from perverted nutrition. This is mainly the result of faults of diet, and consists in a deficiency of certain elements in food. It can as certainly, though more slowly, be cured by the addition of such elements to the food as scurvy can be cured by the addition of antiscorbutics. The chief and constant defect appears to be an insufficient supply of animal fat, and therewith also, in certain cases, a deficiency of earthy salts in the form of phosphates; at the same time, if animal proteid be deficient the disease is intensified. The development of the disease is also favoured, and it is aggravated in degree by evil external

hygienic conditions, such as want of light, warmth, and pure air. The disease is modified in character by the concurrent existence of congenital syphilis or of scurvy. Lastly, in some cases when the fault of diet is not sufficiently great to cause it unaided and alone, the appearance of the disease is determined by the other factors.

The increased vascularity of the ossifying cartilage, and the excessive proliferation of cells observed in rickety bone, is suggestive of a subacute or chronic inflammatory condition; and this view is supported by the experiment of Kassowitz, who produced a state of bone corresponding to that of rickets by inducing hyperæmia in the limb of a growing animal by means of repeated applications of an Esmarch's bandage. The increased fibrosis and cell proliferation observed in the liver and spleen in certain cases is consistent with this observation, although it is to be noted that these changes have not been found in all instances or in other tissues undoubtedly affected by the rickety condition. The agent in human rickets has been referred to some irritant derivative of food circulating in the blood. It has been shown that lactic acid derived from carbohydrate food cannot be regarded as the peccant matter; but it may possibly be something formed from altered materials present in excess, because unused in the disorganised condition of the formative process. The profuse sweatings are suggestive of a like cause; while on the other hand the absence of pyrexia seems opposed to this view, although not conclusive against it. It is possible that the abnormal vascularity and cell proliferation are the results of the impetus of the formative process left unsatisfied in its normal direction for want of materials for the building of the permanent structure.

Diagnosis.—There is no difficulty in recognising the existence of rickets when it is well marked. The bone changes and other symptoms previously described are characteristic and unmistakable. In mild cases of the disease, however, when the signs are less pronounced, the condition may be overlooked, and is, as a matter of fact, constantly overlooked; such cases being put down as mere weakness, debility, or anæmia. In these less pronounced cases there may be nothing beyond beading of the ribs, slight enlargement of the wrists, squareness of forehead, too widely open a fontanelle, backwardness in teething, early decay of teeth, head sweats at night, feebleness of muscle or laxity of joints, and perhaps laryngismus. The occurrence of any one of these signs should excite suspicion, and should lead to a minute examination of other points; a concurrence of several of them would establish the existence of the disease. It must be remembered, however, that rickets may affect one part of the body in pronounced degree, whilst in the other parts the changes may be relatively slight.

With the exception of this mistake of regarding a case of slight rickets as a mere condition of simple debility and anæmia, the most common error, perhaps, is that of mistaking the severe form with great muscular debility for the paraplegic form of infantile paralysis. Rickets, however, is usually distinguished by the fact that although the child may

be quite unable to stand, it is able to use its legs freely; and although in those cases where there is scurvy in addition to the rickets the limbs are kept motionless, the marked tenderness, the dread of movement, the spongy gums, the periosteal swelling or other signs, serve to reveal the true nature of the apparent paralysis.

Prognosis.—With regard to the rhachitic state itself the prognosis is favourable if the patient be placed under suitable conditions. The tendency is towards recovery; the chief question is the degree of bony deformity or stunting of structure which will remain. This will depend partly upon the acuteness and severity of the affection, partly upon the stage at which it comes under treatment. Rickets is not a fatal disease in itself, yet it is liable to aberrations and complications which endanger life. The child suffering from rickets is in a state of constant insecurity. Not only is there the special tendency to pulmonary catarrh, but the danger of this is greatly aggravated by the soft condition of the bony framework of the thorax, which prevents full inflation, especially under obstructive difficulty; while the deformity of the chest favours pulmonary collapse. Accordingly all diseases of which bronchitis is a common complication—such as measles or whooping-cough—become far more grave if the child be rickety. Similarly the tendency to catarrh of the intestine adds to the frequency and severity of attacks of diarrhœa; the instability of the reflex nervous system gives rise to laryngismus stridulus and convulsions, which may prove fatal; while the supervention of scurvy or splenic anæmia adds much to the gravity of the condition and to the uncertainty of the prognosis.

Treatment.—*Prevention.*—Rickets in its ordinary form, being essentially a diet disease, although frequently aggravated or intensified by external injurious influences, is eminently a preventible disease. It cannot, indeed, be stamped out until poverty is stamped out; rickety diet is cheaper and less troublesome than a non-rickety diet. In great cities especially, where the disease is most rife, milk is dear and often largely deprived of cream, while the other factors of defective health conditions prevail there likewise. The disease can, however, be absolutely prevented in most cases, and the fact that it is abundantly found amongst the children of well-to-do people, although it is less common and less severe in this class than amongst the poor, is a grave reflection upon those responsible for their nurture. Rickets, as I have shown, has its origin almost invariably in certain errors in bringing up by hand—either (i.) in the use of artificial foods which are deficient in the most important elements of structural growth, or (ii.) in the administration of foods which, although they contain the essential elements in sufficient quantity, contain some of them in a form not easily digested. In cows' milk, for example, all the essential elements are present, but the caseine is liable to set up gastro-intestinal disorder with vomiting and diarrhœa; thus the elements in question, although present in ample quantity in the food, are drained away undigested and unused. In the majority of instances the two faults are combined: in order to avoid the recurrence

of the disturbance which the latter excited, a diet deficient in the necessary elements is substituted for the sufficiently rich but indigestible one. Thus the morbid state initiated by the first error is accentuated and increased by the means taken to repair it. The great point in the prevention of rickets, especially if the child has to be weaned soon after birth, is to take the utmost care not only to give a food which is properly nutritive, but one which will not by its indigestibility set up gastro-intestinal trouble. It must contain animal fat, proteid, carbohydrates, and salts in the proportions which obtain in human milk—that is (roughly) $2\frac{1}{2}$, $2\frac{1}{2}$, 6, ·34 per cent respectively—in easily digestible form. The proportion in which these elements exist in any given food can be easily estimated by consulting the tables of analyses which show the various constituents. The safest method is to transfer the infant to a good wet nurse; but this is an expedient open only to the wealthy, and, even if expense be not a bar, it is not always easy to secure a satisfactory foster-mother. The next best plan is to feed the child on asses' milk, which closely resembles human milk in composition. This should be scalded or raised to the boiling-point as soon as received in order to prevent contamination, and passed through fine muslin to remove the small coagula which occasionally form on heating. Here again, however, the difficulty of expense and supply is often prohibitive. Failing these measures, a mixture of boiled cows' milk and barley water may be tried, beginning with 1 part of milk to 4 of water, and increasing the strength as the stomach can bear it. If boiled cows' milk, simply diluted thus, produce any gastric disturbance, pancreatised milk diluted with an equal quantity of water should be substituted. After a time the degree of predigestion should be very gradually lessened, by reducing both the pancreatising agent and the length of time allowed for the process, until eventually it is discontinued altogether. This end should be attained in the course of a month or six weeks if possible. If the pancreatised milk be too long continued the function of the stomach becomes seriously enfeebled, nutrition is impaired, and a store of digestive troubles laid up for the future. The same caution holds good with regard to all the predigested foods, patent or other, as well as to peptonised milk; their continued use is eventually injurious.

The proportion of milk to water may be increased until, by the age of six months, that of two to one is reached. If the child be not able to digest cows' milk, except when so diluted that it cannot be taken in quantity sufficient to supply the necessary nourishment, and if asses' milk or a wet nurse be not available, raw meat juice and cream may be added to it or substituted for it. In cases where a moderate amount of curd can be digested, the so-called humanised milk, made by removing a portion of the curd by rennet, may be used. It should be freshly made, and, at first, diluted with one-third water. It ought not to be continued beyond the age of three months, since after that age a larger amount of proteid is required, and the child is liable to fail in nutrition; cows' milk and water should then be gradually substituted by admixture with

it in increasing quantity. It is important to ascertain that the milk contains a full proportion of cream, namely, 2½ to 4 per cent.

In the early months of infancy farinaceous food should only be given when it is necessary as a supplement to fresh milk, or its equivalent of fresh animal material. The malted forms are to be preferred since they add soluble carbohydrate, namely, dextrine and maltose, as well as the soluble proteid gluten. Further, no artificial food of any kind should be regarded as sufficient in itself alone; fresh milk should be added, or its equivalent of fresh animal constituents.

At six to eight months, when the child is brought up by hand, a malted farinaceous food may form a regular addition to the milk and water; or the finest entire wheat flour, or fine oatmeal, may be used if they are thoroughly boiled and the coarser particles strained off. This should be gradually changed to boiled bread and milk, which by the time the child is twelve months old should form the staple food. At ten or twelve months the diet may be increased by the addition of a cup of chicken or beef tea, with a scalded rusk, for the mid-day meal; and the nutritive properties of the meat tea may be further improved by boiling in it some simple vegetables, such as potato and carrot, and straining them out before use. Between twelve and eighteen months a little bread and butter may be given, and the yolk of a lightly-boiled egg; the mid-day meal may be advanced to well-boiled white fish, or the soft pulp scraped off a slice of underdone mutton, with potato thoroughly mashed and rubbed through a sieve and mixed with gravy. As the age of two years is approached—or even earlier—at eighteen months, if the child is robust—a milk pudding or custard pudding or a well-baked apple may be given at dinner in addition; a little red-currant jelly with bread and butter, or a sponge cake, makes a suitable addition to milk at tea-time.

Correct feeding on a diet of this kind—namely, fresh food containing all the essential elements in due proportion in a form which a child can readily digest, so that it produces no gastric disturbance—is the chief point in the prevention of rickets. But in addition to this the other canons of healthy existence must be observed also. Sunshine and light, so far as they may be obtainable in this climate, are powerful agents in aiding vital processes, and are of immense importance together with fresh air, large, well-ventilated sleeping-rooms, and warm clothing to body and limbs. These essentials to full vigour of growth are very imperfectly secured even amongst the higher class of people. Nurseries are frequently overcrowded, and not infrequently, for the sake of convenience, the poorest rooms are assigned for the purpose. Arms, legs, and necks are left bare, and light linen clothing used when warm woollen materials are required.

Curative treatment.—As in the case of prevention so in the cure of rickets, treatment is essentially hygienic and chiefly dietetic. Drugs play a secondary part. As a rule far too much reliance is placed upon them, and children are drenched with cod-liver oil, iron, "chemical food,"

or lime water, often to the disturbance of digestion and the impairment of natural appetite, and consequently of nutrition. Such remedies are useful and have their place; but they are by no means essential or of prime importance in most instances. Drugs are in reality chiefly useful in the treatment of complications.

As explained in speaking of prevention, the position is usually as follows:—The child has originally been placed upon a diet which has set up gastro-intestinal catarrh from the irritation of imperfectly digested fermenting material, such as the massive curd clot of cows' milk; this leaves behind it a chronic digestive inability, so that diluted milk, of sufficient strength to supply the required nutriment, sets up flatulence, vomiting, colic, and diarrhœa. In order to obviate this some artificial food has been given in place of it, which is deficient in the nutritive essentials; or a dozen different foods have been tried one after the other in blind experiment, with the result of perpetuating the disorder and increasing malnutrition. In other cases a food of imperfect nutritive quality, deficient in fat and proteid, has been given from the first; this may agree perfectly perhaps with the digestion, but be eminently productive of the rickety condition; that is, a rhachitic diet. The great object is so to amend the diet that it shall yield proteid and fat in digestible form up to the full standard.

The diet to be adopted must be on the lines previously laid down for the prevention of the disease. It may chance, however, that the child is unable to digest the nourishment proper for its age; as, for example, the common aliment of cows' milk and water of strength sufficient for growth and nutrition. In this case the deficiency of fat must be made up by the addition of cream; and the cream should be continuously and gradually increased from a few drops to a dessertspoonful in each bottle if required, the quantity being regulated according to the age of the child, its power of digesting it, and the amount required to raise the food to the full standard of 3 to 4 per cent. The proteid is best increased by the addition of raw meat juice; this is the most easily assimilated of all proteid matter, and has the additional advantage of being the most powerful of all remedies for anæmia; probably by virtue of the iron of the fresh hæmoglobin. Raw meat juice should be prepared by soaking finely minced beef in an equal quantity of cold water for half an hour, and expressing all juice through fine muslin by twisting it. A teaspoonful of this should be added to the bottle for a young child; but as it does not keep well, and must be freshly made each time, it may be given once a day in the larger quantity of a dessertspoonful up to two tablespoonfuls according to the age and requirements of the individual patient.

In the case of the poor, cream, or even good milk containing a due proportion of cream, is out of reach, and cod-liver oil may have to be given in its place. With older children fat boiled bacon, or the liquid fat of broiled bacon, forms a most digestible and satisfactory substitute. If the child is wasted as well as flabby and bloodless, some form of

malted food, preferably one made from entire wheat flour, should be added to the milk. In the majority of cases of rickets, even if the diet be such as to comprise the standard elements in correct proportion, an increase in the amount of assimilable fat and proteid in the shape of cream or cod-liver oil and raw meat juice will greatly quicken the rate of recovery, care being taken not to overtax the child's digestive powers.

Finally, in cases where the limbs are so tender as to give rise to a suspicion of incipient scurvy, and indeed in all cases where there is marked feebleness and anæmia, it is well to give some fresh vegetable juices. This is best done for young children in the way previously described by boiling potato and carrots in their meat, teas, or broths, and straining them. To children of eighteen months or more these vegetables themselves may be given, if well boiled and thoroughly mashed.

Drugs.—It has been already affirmed that drugs play a secondary part in the treatment of rickets. Cod-liver oil is useful in some cases, especially when good milk and cream are not attainable, or not well borne, but it is to be regarded rather as a food than as a drug; so far as remedial power is concerned it appears to possess no advantage over other animal fats, such as those of cream and bacon.

Cod-liver oil is frequently given in too large doses, so that it interferes with digestion, or sets up diarrhœa. It is to be remembered that a rickety child is prone to gastro-intestinal catarrh, and one teaspoonful of oil twice a day in addition to proper diet is sufficient for a child two years old. The oil may be rendered less laxative by the addition of an equal quantity of lime water, with which it forms an emulsion. If, however, the cod-liver oil cause undue looseness of the bowels, it does harm rather than good, and should be discontinued. The intestinal flux interferes with the absorption of nutriment, and drains it away. Iron is useful for the relief of anæmia, and may be given with cod-liver oil in the form of steel wine, or syrup of the phosphate, or citrate, although these preparations of iron are less effective than raw meat juice as a means of restoring red blood corpuscles and their hæmoglobin. The earthy phosphates are present in ample quantity in milk, and in the farinaceous preparations made from wheaten flour or oats; and they are probably most easily assimilated when administered in food. In some cases of more extreme disease, or where food is taken in insufficient quantity, it may be well to give lime salts in the form of the syrup of lactophosphate of lime, or of the so-called chemical food. Experience shows that lime water is quite useless in this respect, and that the salt must be in the form of phosphate.

In some instances the bitter tonics, such as quinine and bark, are useful for the purpose of giving tone and appetite; but it is doubtful if their influence for good compensates the drawbacks which attend their administration to young infants. Iodide of iron, so frequently given, is, in my experience, of no service, and, indeed, harmful from its depressant and blood-deteriorating properties; when given to children for a long time in frequent doses it has been observed to produce purpura and

great debility; indeed iodides in any form are clearly contra-indicated in a disease of degraded nutrition like rickets.

Phosphorus, on the strength of its reputed power in consolidating new formations of bone, was adopted by Kassowitz as a remedy for rickets; and it has been used to a considerable extent by continental physicians, with results which are regarded by the majority of them as highly favourable.

Phosphorus must be given dissolved in olive oil, and in extremely small doses, beginning with $\frac{1}{125}$ grain, in order to avoid gastro-intestinal catarrh, since it acts as a local irritant of the mucous membrane. Of the value of this treatment I have had no experience. The results which follow correct dietetic treatment, aided where possible by other hygienic conditions, are so satisfactory that I have found no further measures necessary. In certain cases of extreme severity, perhaps, this additional agent, although directed solely to the relief of bone defect, may be of service in pushing forward repair; it is in such cases, however, that the drug is constantly prohibited by the catarrhal inflammatory condition of the alimentary canal which almost invariably coexists.

Massage is of great service in the treatment of rickets. It improves the nutrition of tissue by increasing the flow of blood in the parts, thus aiding the advent of fresh nutritive material; it also hastens the removal of effete products by quickening the circulation. The practice of massage is described in the article devoted to this subject [vol. i. p. 373].

In cases where the condition borders on scurvy, and there is tenderness of the limbs from persistent engorgement, massage is not available.

Baths again are of service in improving the circulation, aiding nutrition, and giving tone to the nervous system. The best method, after the child reaches the age of six months, is to allow it to stand in a warm bath of 90° to 98°, and to apply a douche of colder water at 60° to 70° freely with a sponge for a few seconds. For children of ten or twelve months salt water may be substituted for fresh water. The advantage of allowing the child to sit or stand in a warm bath while douched with cooler water is the avoidance of chill, usually shown by cold hands and feet, which is readily produced in rickety children; many weakly infants turn blue in a bath of 70° or 80°, and then the bath does far more harm than good. In all cases the bath should be followed by steady friction before the fire, until surface warmth and circulation are fully restored.

Clothing.—As the temperature of the body in rickets, when no complications are present, is persistently subnormal, the preservation of body heat by appropriate clothing is an important element in the treatment. Not only is temperature below normal, but the heart muscle is feeble, arterial tone impaired, and the general circulation depressed. Thus the child suffers from coldness of the extremities, and is easily affected by surface chill; the cooling process is further favoured by the evaporation of the profuse perspiration, whereby congestion of internal organs is readily produced. The condition of the growing bones is also unfavourably influenced by coldness of the limbs. The underclothing should be

of soft woollen material, the arms and neck being kept covered even indoors by long sleeves and high-necked dresses, and the legs by drawers or leggings. The only modification on going out of doors, or for change of season, should be in the matter of the thickness, not in the nature of the material. A night-dress of fine flannel is advisable, so that lighter bed-clothes may be used, and the chilling avoided which is apt to result when the child throws them off.

Fresh Air.—The value of abundance of fresh air in maintaining the health of children is very generally acknowledged—but practice falls greatly behind principle in this respect. Delicate children, especially those with a tendency to catarrhs, and rickety children, are often kept too closely indoors, and the temperature of the rooms and house in which they live is kept too high. The chief reason why the child "takes cold" in such cases is the change from the warm atmosphere of the room to the colder air outside; the skin and mucous membrane being morbidly hypersensitive. The way to remedy this instability and increase their powers of resistance to the reflex disturbances of cold and damp in this chill climate, is not to confine the child to warm rooms; nor to heat halls and passages with hot-water pipes and stoves, but, under ordinary circumstances, to keep down the temperature of the nursery to the reasonable standard of 60°, or even a few degrees lower. Delicate children of all kinds, and rickety children amongst them, may be taken out of doors even in somewhat severe weather for very short periods, which may be repeated, always provided that great care be taken to keep up the body heat by warm clothing, shelter from the wind, and the administration of a little food before going out. Another point of equal or even greater importance in the treatment of rickets is the ventilation of the rooms, and especially of the sleeping-rooms. As mentioned in treating of prevention, nurseries are frequently greatly overcrowded and badly ventilated. In the case of a rickety child the cubic space allowed should be ample or even excessive—800 to 1000 cubic feet, and the room should be warmed by a good open fire or fires, so as to assist ventilation by a free out-draught, and not by stoves or hot pipes in any form, which injuriously affect the quality of the air. No nursery should be lighted by gas, not merely for the negative reason that it consumes oxygen largely, but because the products of combustion which are given off are positively injurious. Candles and lamps burning pure vegetable oil, which consume less air and give off little except simple carbonic acid and water, are comparatively harmless. The electric light, which consumes no air and gives off no products of combustion, is the best of all. The ventilation of the nurseries by the usual means of window, door, and chimney should be aided by Tobin's tubes. Further, if the child live in a town, or in a place in the country which is damp and cold, or much shut in by trees, it should be removed to a fresh open situation, with dry soil and bright atmosphere; and near the sea if possible. It is desirable that it should remain away from home until the acuter signs of the malady have entirely disappeared.

Light and Sunshine.—The sun is the source of all energy, and its rays are essential to the life and growth of the higher animal and vegetable organisms. The powerful influence which privation of light and of the direct rays of the sun has upon the production of rickets is evidenced, as previously stated, by the greater prevalence of the disease in the clouded skies of temperate climates, and especially by the presence of its severer forms in the added darkness of great cities. The influence of sunlight in prevention is shown by the rarity of the disease in the sunny climates of the south. The effect of sunlight is probably as potent or more potent than that of fresh air. Light, and especially full sunlight, is as important to the cure as to the prevention of rickets, and the removal of a child thus suffering to a bright and sunny climate is of great value in hastening its recovery.

The treatment of *deformities* comes largely into the province of the surgeon, and it is not necessary to speak of it here at any length. It may, however, be pointed out, first, that care during the early stage does much to obviate deformity; and, secondly, that the deformities tend to get well. If the bending be not great in degree many bent bones gradually become straight. If the attack be severe the horizontal posture should be maintained, and the back and limbs be duly and evenly supported and kept at rest, especially if the parts are tender. Partial fractures and acute bending should be treated on the same principle under skilled surgical direction. The use of splints during the early stage is of little value, if any, for the purpose of straightening curvatures or lax joints; but a long splint protruding beyond the foot is of service in some cases to prevent walking. If there be no tenderness of bone, massage is of great value in improving the nutrition of bone as well as of muscle and tendon, and in lessening the ultimate deformity.

Treatment of concurrent disorders. — Gastro-intestinal disorders are common, and are of importance as leading to malnutrition in two ways; namely, by imperfect digestion and absorption, and by the draining away of nutriment by means of vomiting and diarrhœa.

Here again the main point in treatment is a careful regulation of diet. Inability to digest the caseine of cows' milk is the most frequent direct cause of gastro-intestinal disorder in young infants. The cows' milk must be stopped at once and human milk, or asses' milk, substituted, if possible; or the milk may be pancreatised for a time and diluted—the pancreatisation being gradually reduced; or cream and water may be given, beginning with a strength of 1 teaspoonful to 3 ounces, and increasing it to 1 teaspoonful to 2 ounces. Freshly-expressed raw meat juice may be added to this mixture in the proportion of 1 teaspoonful to 4 ounces; or chicken tea or mutton tea or beef tea given independently as a supplement. If the child is eight to twelve months old, some farinaceous preparation, such as one of the malted foods or bread previously well scalded with boiling water, may be added to the milk or its substitute. The dieting should indeed be conducted upon the general plan laid down beforehand with respect to the prevention

and cure of the main condition. If the vomiting and diarrhœa are severe, food of the blandest kind, such as thin bread, jelly food, weak chicken tea, dilute peptonised milk, or scalded asses' milk, should be given in small quantities of half an ounce to an ounce at a time, at intervals of an hour. If collapse be present, brandy, in doses of 5 to 20 drops in a tea- or dessertspoonful of water, should be given every four hours. Valentine's juice in doses of 10 to 20 drops in a tablespoonful of water is often retained when other foods are rejected, and serves to tide over a crisis.

If the stools contain much undigested matter, such as curdy masses, and are slimy and offensive, medicinal treatment may well begin with a small dose of castor oil—diluted with an equal quantity of olive oil in the case of young infants—to clear the noxious matter away. This should be followed by a few doses of gray powder guarded by Dover's powder; a sixth to half a grain of each may be given every four hours to a child six months old. If the bowels are at all loose, so that three or more watery evacuations are passed in twenty-four hours, the great point, next to giving fluid diet in small quantities, is to get the diarrhœa under control at the earliest moment by full doses of subnitrate of bismuth —10 grains in 2 drachms of water may be given every four hours to a child six months old. The small doses of 2 or 3 grains usually prescribed are quite ineffectual; and the soluble preparations of the drug are equally so. The bismuth is best given with spirit of chloroform (♏j. to ♏ij.) and syrup (♏xv. to each dose). If the peristalsis be very active, and the discharge from the bowels copious and frequent, the addition of opium is essential. It should be given in the form of nepenthe or liq. opii sedativus, in doses of a quarter to half a drop for a child six months old. Small injections of starch, with or without a drop of tincture of opium, are useful in lessening the irritability of the rectum. If the colon be specially implicated, as shown by the passage of mucus and blood, simple injections of warm water are useful to wash out the bowel before the administration of the starch enema; they also aid the effect of the bismuth.

Pulmonary disorders.—Bronchitis, Bronchopneumonia, Collapse.—The treatment of these complications should proceed on the ordinary lines adopted in such conditions. It is well, however, to call attention to, and to emphasise the fact that all depressing remedies—such as antimony, aconite, antipyrin, and the so-called antipyretics generally—are badly borne by rickety children, and should be strictly avoided; also that the cure of the rickety state itself by correct dieting should proceed as far as practicable step by step with that of the immediate pulmonary trouble, in order to prevent the recurrence of such disorders in the future.

Nervous disorders—Laryngismus, Convulsions, and Tetany—which arise out of the rickety state involve a certain risk to life. In these cases remedial measures must be carried out on three distinct lines concurrently.

i. To remove any source of irritation which may be a cause of reflex disturbance. This is found most frequently in the alimentary

canal in the form of undigested food, or of an irritated catarrhal condition of the mucous membrane with consecutive diarrhœa. A dose of castor oil, followed by gray and Dover's powder, or bismuth and opium as previously prescribed, with proper dieting, will usually correct the disturbance. Another common source of reflex irritation of the cord, sufficient to produce convulsions in a rickety child, is that of teething; a timely use of the gum lancet, now too little as formerly it was too frequently and unreasonably used, will often arrest a threatened attack.

ii. To soothe the abnormal excitability of the central nervous system, and keep it dulled until such time as the source of irritation is removed, and the stability of the centres established. For this purpose chloral has more power than any other drug; its efficiency appears to be increased by combination with the bromide of potassium or ammonium. The use of sedatives has been decried, but they are of signal service in enabling the patient to tide over the period of most active danger. To a child six months old chloral may be given in doses of half a grain to a grain, with 3 or 4 grains of bromide of ammonium, every four hours, until the attacks of laryngismus, the carpopedal contractions, or the general convulsions are effectually controlled: the bromide must be diminished or omitted after a time in order to avoid exciting the well-known pustular eruption of the skin; but the chloral may be continued as long as necessary, if care be taken to reduce or stop it if undue drowsiness appear.

The attacks of laryngismus and general convulsions are usually completely arrested after a short time by chloral and bromide, if given in sufficient doses and the patient be brought fully under their influence. If convulsions are so severe that the mixture cannot be taken by the mouth, the same drugs may be given by rectum in larger doses as an enema; namely, 2 to 3 grains of chloral and 5 to 10 of bromide of ammonium (according to age) in half an ounce of water. This measure is even more effectual and more rapid in action than administration by the mouth. The actual attack of laryngismus, which is occasionally fatal, may be arrested by dashing cold water on the face.

iii. The third point in treatment is, as before, to proceed with the cure of the rickety state by the appropriate diet and regimen laid down in speaking of the general treatment of the disease; thus we shall remove the fundamental fault which lies beneath all the various manifestations of it which have been described.

W. B. CHEADLE.

REFERENCES

1. ACKER, G. N. *Arch. Pediat. N.Y.* 1894, vol. xi. pp. 893-898.—2. ANDREW. *Australasian Med. Gaz.* 1892, vol. xi. p. 418.—3. BAGINSKY. *Lehrbuch d. Kinderkrank.* (Wreden's Sammlung), B. vi. p. 270 *et seq.* (1883).—4. BARLOW. Bradshaw Lecture, *Brit. Med. Journ.* 1894, ii. p. 1029.—5. BARLOW and BURY. Keating's *Cyc. Chil. Dis.* vol. ii.—6. BARLOW and LEES. *Path. Trans.* vol. xxxii. p. 223 *et seq.*—7. BAXTER. *St. Bart. Hosp. Rep.* vol. iv. p. 78.—8. BOODLE. *Australasian Med. Gaz.* 1892, vol. xi. p. 140.—9. BOWLBY, A. A. *Surgical Pathology*, p. 339.—10. CANTLEY, E. Recru-

descent or late Rickets," *Brit. Med. Journ.* Jan. 4, 1896.—11. CHEADLE. *Brit. Med. Journ.* 1888, ii. pp. 1145-48 ; *Artificial Feeding of Infants*, 4th edit. 1896, Lect. v. pp. 166-207. —12. COMBY. *Le Rachitisme*, Paris, 1892 : *Bull. et mém. Soc. méd. du hôp. de Par.* 1893, 3. s. x. pp. 486-489 ; *Pratique méd. Par.* 1894, viii. 183.—13. FITZGERALD. *Australian Med. Journ.* 1887, New Series, vol. ix. p. 413.—14. FRIEDLEBEN. *Jahrb. f. Kinderheilkunde*, B. iii. H. ii. pp. 61-137, pp. 147-178.—15. GLISSON. *A Treatise of The Rickets, being a Disease common to Children*, trans. by Phil. Armin, London, 1651 ; *De rachitide, sive morbo puerili*, 1671.—16. GOWERS. *Clinical Journ.* 1894, vol. iv. p. 309.—17. GUÉRIN, J. *Mémoires sur les Difformités du système osseux*, Paris, 1889. —18. HEITZMANN. *Sitzgsber. d. kais. östr. Acad. d. Wiss. in Wien*, 1873, No. 17. —19. HERZFELD. *Centralb. f. Gynäk.* 1894, No. 18.—20. JENNER, Sir W. *Clinical Lectures on Rickets, and other Subjects.* Lond. 1895.—21. KASSOWITZ. Bd. ii. (1882), *Rachitis ;* Bd. ii. Ab. 2 (1885), *Path. der Rachitis ; Méd. inf. Par.* 1894, i. pp. 547-552.—22. KORSAKOFF, N. S. *Med. Obozr. Mosk.* 1892, xxxviii. pp. 314-378.—23. LAURO, V. *Ann. di Ostet.* Milano, 1887, ix. pp. 385-429.—24. MEIGS, J. F., and W. PEPPER. *Practical Treatise on Dis. of Child.* 4th ed. London, 1882.—25. MELTENHEIMER. *Jahrb. für Kinderkrank.* xxxii. 8.—26. MUSKETT. *Australasian Med. Gaz.* 1891, x. pp. 285-291.—27. OWEN, I. Rep. of Collect. Invest. Comm. *Brit. Med. Journ.* 1889, i. p. 114.—28. RAILTON, T. C. *Brit. Med. Journ.* 1894, i. p. 1299.—29. REHN. *Handb. d. Kinderkrank.* (Gerhardt), B. iii. H. 1, p. 40 *et seq.* (1878).—30. SMITH, LEWIS. "Starr's Dis. of Children," *Arch. Pediat. N.Y.* 1893, x. pp. 996-1000.—31. SNOW, IRVING. *Med. News* (Phila.), 1894, lxv. pp. 316-320.—32. SYMINGTON and A. THOMSON. *Lab. Rep. Roy. Coll. Phys.* 1892, vol. iv. p. 238.—33. THOMSON, J. *Edin. Med. Journ.* 1892-3, vol. xxxviii. p. 1109.—34. TOWNSEND, C. W. *Arch. Pediat. N.Y.* 1894, xi. 761-763.—35. TRIPIER, L. *Mém. et compt. rend. soc. de sc. méd. de Lyon*, 1863, iii. pp. 199-208 ; *Arch. de Physiol. norm. et path. Par.* 1874, 2. S. i. pp. 108-125.—36. TURNER, A. J. *Australasian Med. Gaz.* 1892, p. 104.—37. POIT, C. *Sitzgsber. d. math.-phys. Class. der k. b. Akad. d. Wiss. zu München*, Bd. xix. Jahrgang 1889, pp. 437-8.—38. VOIT, E. *Sitzgsber. d. Gesellsch. f. Morphol. u. Physiol. in München*, 1889-90, v. 101. —39. WEGNER. *Berl. klin. Woch.* 1869, No. 39 ; "Vehdl. des Berl. med. Ges." ; *Virchow's Arch.* Bd. 50, H. 3, p. 329 ; *ibid.* Bd. 55, p. 39.—40. WOODHEAD, SIMS. *Practical Pathology*, London, 1892, pp. 469-472.

W. B. C.

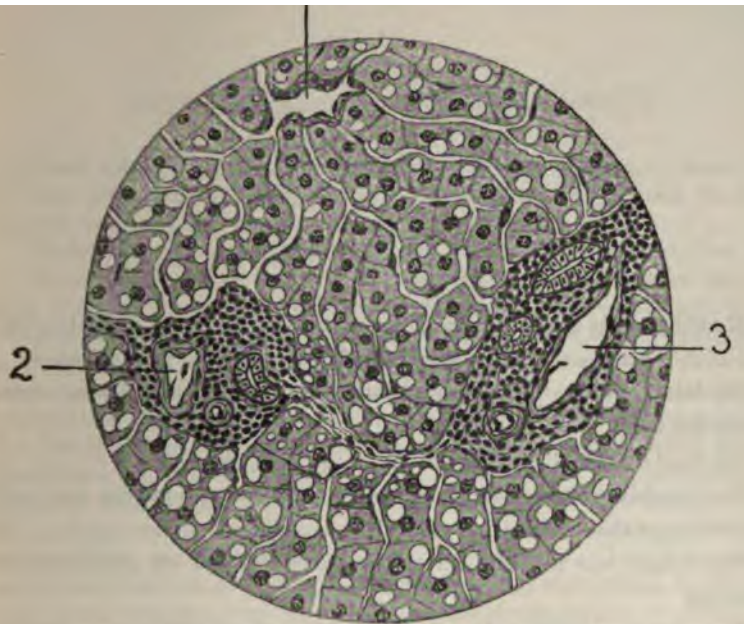


FIG. 1.—Section of liver, high power (Hartnack, oc. 7, obj. 4). 1, An intralobular vein; 2 and 3, Branches of the portal vein: the portal canals in which they lie show a marked proliferation of the connective tissue cells. The liver-cells contain fat-drops in excess of the normal.

FIG. 2.—Section of small intestine including the margin of a Peyer's patch, low power (Hartnack, oc. 3, obj. 4). 1, Lymph-nodule the cells of which are somewhat swollen; 2, A villus from which the epithelium has become detached. The villus is swollen, and, like the rest of the mucosa, contains an excessive amount of lymphoid tissue.

[*For description see over leaf.*

Morbid changes in the viscera in a case of extreme rickets and tetany in a child of thirteen months, which ended fatally from repeated convulsions :—

The brain, spinal cord, and meninges, and posterior tibial nerves were normal, as were the lungs.

The heart showed slight hypertrophy of the right ventricle.

The bronchial and mesenteric glands were twice the natural size, and under the microscope showed a marked general hyperplasia.

The stomach was normal both to the naked eye and on microscopic examination.

The spleen was slightly enlarged, firmer than normal, that is, in a condition resembling "cyanotic induration." The Malpighian bodies were increased in size, and the cells somewhat larger than natural.

The kidneys were normal, except for some small uric acid calculi.

The liver and the small intestine showed the most marked hyperplasia. The accompanying figures, reproduced from original drawings by Mr. Jackson Clarke, represent the minute changes found in these organs.

OSTEOMALACIA, OR MOLLITIES OSSIUM

Mollities Ossium, or **Osteomalacia**, is a disease in which there is a gradual softening and subsequent bending of the bones, and in which spontaneous fractures are very liable to occur.

Pathology.—An examination of the affected bones shows that they are much lighter than natural, and so soft that they may be indented with the fingers or bent by the hands. They are readily cut with a knife, and the section shows that the medullary canal is greatly increased in diameter, and has extended into the epiphyseal ends. In advanced cases the compact tissue is reduced to a mere shell. Microscopical examination reveals an absorption of the lime salts, beginning around the Haversian canals and the canaliculi, such as occurs when bone is placed in a solution of hydrochloric acid; thus while the lime salts are dissolved the animal matrix remains. In this matrix changes ensue of a degenerative nature, which end in a complete gelatiniform degeneration, and the formation of a jelly-like mass. In this process of destruction there is no sign of inflammation; but the vessels, deprived of this normal support, very frequently permit of exudations of blood, and the osteoclasts are much increased in number. A microscopical examination of the medulla and of the decalcified bone shows many blood-cells in various stages of disintegration, with cells of various shapes and sizes and granules of oil, fat, and gelatinous matter.

Ætiology.—No definite cause for mollities ossium can be assigned; but it has been stated that the absorption of bone is due to an excess of lactic acid in the blood, though this excess has not been clearly demonstrated. When occurring during pregnancy the disease has been attributed to the demands of the fœtus for bone salts, but this supposition does not apply to the cases which occur independently of pregnancy. It may be stated that in many cases some causes of nervous or mental depression appear to have been present; but how such antecedents could lead to or bring about the changes described is beyond our comprehension. Mollities ossium occurs with far greater frequency in women than in men. It usually begins between the ages of 25 and 45, and its onset is frequently associated with pregnancy. More rarely it begins about the age of puberty, or even in childhood.

Symptoms.—Before any definite changes are noticed in the bones so-called "rheumatic" pains usually occur, and these may be severe; at the same time the general condition is one of feebleness or debility. The next change to be noticed is a sense of weakness and insecurity in the lower extremities, so that the patient is unwilling to walk without support, and is unable to walk with freedom; the gait is often peculiar, and the steps

are short and unsteady. After this it may be noticed that one or more of the bones are becoming curved, so that the stature of the patient is diminished; whilst in other cases nothing so definite is observed until a bone is fractured by some trivial accident, or until parturition is impeded by deformity of the pelvis.

In advanced cases the deformity may be very great, and it may affect the whole skeleton. The spine, by a great increase of its normal curves, assumes somewhat of the shape of the letter S. The ribs are flattened or even bent inwards on the lateral aspects of the chest, whilst near to the sternum and close to the spine they are protruded or fractured. The sternum is usually pushed forwards, and is often fractured at the juncture of its several portions. The pelvis is most seriously affected, so that parturition may become impossible. The sacrum is pushed downwards by the weight of the body, whilst the acetabula are pressed inwards by the heads of the femurs; thus the pelvic brim assumes a trifoliate form, with the pubic symphysis thrust forward like a beak. The bones of the extremities are bent or fractured in various degrees, and the whole of the limbs may become quite flaccid and useless. The urine contains an excess of lime salts.

The viscera are not affected except as a direct result of the osseous lesions; but, in consequence of the latter, pulmonary congestion and enfeebled respiration are of common occurrence, and an attack of bronchial catarrh may at any time prove rapidly fatal. It should be remembered that the course run by different cases varies very greatly in rate, and that while in some patients all the changes enumerated may follow each other in the course of a year or a little more, in others the affection is limited to the pelvis and sacrum, and appears to progress only during pregnancy; either remaining stationary at other times, or making hardly perceptible progress. Some patients thus live for many years after the onset of the disease, and do not become bedridden at any time.

Treatment.—There is no specific treatment for mollities ossium, but good food and a general tonic line of treatment are indicated. Fractures in the early stage of the disease generally unite with proper care, although they do so slowly.

ANTHONY A. BOWLBY.

OSTEITIS DEFORMANS

Osteitis Deformans was first described by Sir James Paget in the year 1877, and since that time many examples of the disease have been recorded.

It may be defined as a very chronic inflammation of bone occurring in people past middle age, implicating many bones, and accompanied by a peculiar softening and bending of the osseous structure. In a few cases a single bone only is diseased.

It should be noted that in osteitis deformans there is no implication of the general health and no affection of the viscera. The onset is quite insidious, and it is not until some definite deformity of a limb or a considerable enlargement of the head attracts attention that any notice is taken of the bony thickening.

Pathology.—The bones affected by this disease become gradually thickened by the deposit of new bone from the periosteum, and by the same process their normal outlines are slowly obliterated. By the exudation of inflammatory products within the bone the osseous structure is absorbed, rarefied, and softened; the spaces thus formed are filled with inflammatory exudation. The whole bone becomes greatly increased in circumference, and, on section, is seen to be much thickened. In some cases the medullary canal is increased in size.

In consequence of the softening which accompanies the inflammatory process the bones become bent, the normal curves being at first increased; and, after a time, fresh curvatures are established.

The skull is increased in thickness, the forehead becomes large and prominent, and the face, in consequence, appears to be too small for the cranium by which it is overshadowed. The clavicles become much curved and thickened. The thorax falls in on account of the yielding of the softened ribs, and the abdomen becomes prominent. The femurs curve chiefly outwards, and the tibias forwards. The humerus does not curve so much as most of the long bones, but the radius and ulna curve backwards. In consequence of the bending of the bones of the lower extremity and of the general posterior curvature, which is often met with in the spine, the height of the patient is frequently diminished by several inches. The walk is tottering, and the support of a stick is often necessary. The shoulders fall forward over the chest, and the head protrudes in a very peculiar manner, as if too heavy for the cervical vertebræ. The chin tends to rest upon the sternum, and in order to look up, the patient thrusts it out so that the face is carried on a plane which is considerably anterior to that of the body. The course of the disease is slow, and usually extends over many years. A fatal termination may result from the difficulty of respiration caused by the softened thoracic wall.

Indefinite lesions have been found post-mortem in the spinal cord (1).

Symptoms and diagnosis.—Beyond the deformity already described and some aching pains in the extremities there are no noteworthy symptoms. The diagnosis from acromegaly is given under that disease.

Treatment.—No treatment is known to produce any healing effect on the softening and bending of the bones.

LEONTIASIS OSSEA is the name applied to an overgrowth or hyperostosis of all or some of the cranial or facial bones. The formation of new bone is very slow, and may produce no noticeable deformity for many years; and the change may begin at any period of life, having been observed in childhood in several instances. Both sexes are prone to attack; the causes are unknown. The suggestion that the disease is due to rickets is quite unsupported by the facts observed in most cases. There are no concomitant affections of other parts of the body, and any symptoms that ensue are the direct result of the pressure exercised by the new bone. The superior maxillæ and the various bones entering into the formation of the inner walls of the orbits are not infrequently affected alone, the cranial and lower jaw bones remaining free from disease. In other cases the whole of the cranial and facial bones are involved, but the cranial bones are but seldom attacked alone. In the process of growth the affected bones become greatly increased in density, and the open cancellous framework of the ethmoid becomes converted into dense, compact bone. All cavities, such as the ventrum, the sphenoidal and ethmoidal sinuses, and the bony canals for the supra- and infra-orbital venous, become obliterated and their contents destroyed. In addition, there is considerable formation of new bone on the surfaces, and consequent deformity of the features. The cavities of the orbit, the nose, and the mouth may be encroached upon, and proptosis or blindness may ensue upon the pressure exercised upon the eyeball and the optic nerve. The sense of smell may similarly be lost from occlusion of the nostrils or pressure on the olfactory nerve in the ethmoid bone. A single superior maxilla is sometimes alone affected by hyperostosis, and may present precisely the same appearances as a bone from a case of leontiasis. Such an affection of one upper jaw is an infinitely more common disease than is leontiasis ossea: the latter is indeed a very rare affection. The symptoms caused by leontiasis vary with the position and extent of the new bone. Pain is necessarily very common, and occasionally mastication and speech are interfered with. The deformity is sometimes very great. No treatment can be adopted with any hope of success; but occasionally, where the growth is a limited one, some benefit may result from resection of some portion of the affected bone.

ANTHONY A. BOWLBY.

REFERENCES

1. GILLES DE LA TOURETTE and MARINESCO. *Nouv. Iconographie d. l. Salpêtrière*, No. 4, 1895.—2. PAGET, Sir JAMES. *Trans. Royal Med. and Chir. Soc.* vols. lx. lxv.

A. A. B.

ACROMEGALY

Acromegaly is a disease which has not been recognised until recent years, and is one of which there is yet much to be learnt. It is therefore advisable, in describing such an affection, to remember that whilst there are many definitely ascertained facts which have been accurately observed and noted, there are probably many others as yet unrecorded; and that our knowledge of the affection is very scanty and inaccurate.

Symptoms.—The most obvious characteristics of the disease are enlargements of the hands and feet, with much deformity of the face. The hands are usually more affected than the feet, and are increased in breadth and thickness rather than in length, the soft parts being specially concerned in the change. The legs and forearms are not usually enlarged, but in some of the recorded cases the patients were very muscular. The spine usually presents a posterior curve in the dorsal region, and there may be some lumbar lordosis or lateral curvature. The thorax is flattened from side to side, and moves but little in respiration, the respiratory movements being chiefly abdominal. The skin is thick and often warty, and pendulous parts are specially thickened; the labia majora and clitoris may be greatly hypertrophied. The growth of hair may be increased, and the hair itself may become very long and coarse.

The whole head is often enlarged, and the cranium tends to become lengthened antero-posteriorly; the bones of the face are specially increased in size, and the jaw-bones more than the rest; the lower jaw by its great enlargement frequently projects in front of the upper teeth, and causes much disfigurement. The supraciliary ridges and the tarsal cartilages are much hypertrophied, and the whole nose is greatly increased, its bones, cartilages, and skin all suffering from overgrowth. The ears are not usually so much affected. On account of the alteration in shape of the maxillæ complete closure of the teeth may be impossible. The lips are thickened, and the lower lip tends to protrude and to hang down; the expression of the features may be completely altered.

The youngest patient in whom acromegaly has been observed was fifteen years of age, and the oldest was sixty; but most of the cases have been observed between the ages of twenty and forty. Both men and women are attacked. Headache is almost always complained of, and there is often pain in the eyeballs. The general condition is one of lassitude, and the speech is usually thick and slow. The tongue is large

and flabby, and appears too big for the mouth; the cartilages of the larynx in male patients are sometimes enlarged and thickened.

In addition to the changes in the extremities and face, blindness, with atrophy of the optic disc, loss of sense of smell and of taste, and, in women, cessation of the catamenia, have been commonly observed. Indeed, in cases occurring in women the absence of normal menstruation is constant. Muscular weakness and excessive perspiration have also been observed in connection with acromegaly.

In patients on whom post-mortem examinations have been made, it has been found that the bones of the hands and feet were enlarged, that on all the bones there was a tendency to the exaggeration of normal ridges or tubercles, and that on some of the bones of the extremities, as well as on those of the face, there were osteophytic growths, although the general overgrowth of the whole bone was not seen in the extremities. The sternum is usually thickened, widened, and lengthened, and the ribs and clavicles are also greatly thickened; similar alterations are common in the pelvic bones. The uterus is generally atrophied and the vagina capacious.

The pathology of acromegaly is yet obscure, but it becomes more probable year by year that disease of the pituitary body is a constant factor in this disease; in all recent post-mortem examinations this body has been found greatly hypertrophied, the seat of new growth, or else atrophied and cystic: it may be considered certain that the blindness and headache so often noticed are the direct results of the enlargement and pressure of the diseased pituitary body. In a few cases the thyroid gland has been found diseased, but there is no proof that this gland is in any way causatively associated with acromegaly. [*Vide* art. "Exophthalmic Goitre," vol. iv.]

Diagnosis.—The symptoms of acromegaly have been described in detailing the changes noted in the different parts of the body, and the diagnosis is as a rule quite easy. The thick, heavy lips and broad, deformed face have led to confusion of the disease with myxœdema; and the lethargic condition and thick speech increase the superficial resemblance to this affection. The alterations of the skin and hair, however, are not such as occur in myxœdema, and in the latter affection the mental condition is far more early and seriously altered. In myxœdema also there is no such deformity of the extremities and face as in acromegaly.

Acromegaly may be easily separated from osteitis deformans when it is remembered that in the latter affection the bones of the cranium and the long bones of the extremities are mainly involved, whilst in acromegaly the facial bones and bones of the hands and feet are chiefly affected.

Pseudo-hypertrophic pulmonary arthropathy more closely resembles acromegaly than does any other affection, and the detailed differential diagnosis between these two maladies will be found on page 154.

Treatment.—At present no remedy is known to be of much service,

but improvement has been recorded under the use of iodide of potassium and of thyroid extract; these drugs deserve a further trial.

ANTHONY A. BOWLBY.

REFERENCE

PIERRE MARIE and SOUZA-LEITE. *Essays on Acromegaly.* London, New Sydenham Society, 1891.

A. A. B.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Hypertrophic Pulmonary Arthropathy is the name given by Marie to a condition of the hands and feet with other associated lesions which simulates acromegaly in some respects.

The disease is characterised by great enlargement and deformity of the hands, wrists, feet, and ankles, and occurs in patients who are the subjects of some chronic pulmonary affection; it has been seen in connection with empyæma, new growths of the lungs, phthisis, and chronic bronchitis.

Symptoms.—But few cases have as yet been observed, and those described by W. Thorburn and Rufenacht Walters appear to be the only ones yet recorded in England. There is still very much to be learnt about this malady, and any descriptions now given must be regarded as provisional until further opportunities arise for studying the morbid anatomy of the parts involved. The character of the enlargement of the hands is peculiar to the disease, for, whilst the carpal and metacarpal regions are but little widened and lengthened, the fingers are much increased both in length and in thickness, the terminal phalanx especially being clubbed and enlarged, and the nail greatly curved, striated, and fibrous, so that it resembles the talon of a bird. The wrist-joint is swollen, and the carpal extremities of the radius and ulna are increased in size. Such a hand differs from that of acromegaly in several respects, for in the latter affection (*a*) the nails are not altered and the finger-ends are not clubbed; (*b*) the fingers are not much increased in length; (*c*) the carpus and metacarpus are greatly widened.

The changes in the feet are very similar to those in the hands: the toes and their nails are much lengthened; the tarsus is little involved, but the ankle-joint and articular ends of the tibia and fibula are swollen.

In addition to the changes in the hands and feet there is sometimes swelling of the articular ends of various long bones, and in most cases distinct effusion into the knee-joints has been found. In at least half of the cases there has been spinal curvature in the dorso-lumbar region, and in some a very definite angular curve resembling that of spinal caries.

It is said that in the earlier stages, corresponding to an alleged decalcification of bone, the lime in the urine may reach double the normal standard; the phosphoric acid is extremely low, and urea the minimum normal (2).

The diagnosis of this affection from acromegaly is quite easy to make, for there is no deformity of the face, and none of the associated symptoms referable to the sight, the speech, the menstrual and other functions, which occur when acromegaly is present; moreover, as already pointed out, the character of the enlargement of the hands and feet differs in the two affections. In either case there may be a spinal curvature, but in acromegaly the curve is in the upper dorsal or cervical region; in the malady before us it is lower down, and in acromegaly there is never the angular projection which may be seen in hypertrophic pulmonary osteo-arthropathy.

Pathology.—Few autopsies have been obtained in this disease, and its pathology is at present quite obscure. In the three recorded post-mortem examinations the bones of the affected parts were found thickened with periosteal new bone, and in one case they were the seat of rarefactive osteitis. In the remaining case (that of Rauzier) the synovial fluid was increased, and the cartilages of the knees and elbow were eroded. In all three cases the extra-articular soft tissues appeared unaffected. One necropsy only has as yet been recorded in England (6).

The disease is attributed by Marie to absorption of some toxic material from the diseased pulmonary tissues; Thorburn, however, considers the lesions tubercular, and suggests the name "Tubercular poly-arthritis."

ANTHONY A. BOWLBY.

REFERENCES

1. *Gaz. des hôpitaux*, 1892, p. 685.—2. GUÉRIN and ÉTIENNE. *Arch. de méd. expér.* July 1896.—3. MARIE and SOUZA-LEITE. *Loc. cit.*—4. *Revue de médecine*, 1890, p. 361; 1891, p. 30; 1892, p. 231.—5. THORBURN. *Brit. Med. Journal*, June 3, 1893. —6. THORBURN and WESTMACOTT. (Necropsy.) *Path. Soc. Trans.* vol. xlvii. p. 177.

A. A. B.

GOUT

SYNONYMS.—*Podagra; Arthritis Uratica.* Latin, *Gutta;* French, *Goutte;* German, *Gicht.*

THE name Gout is derived from the Latin *gutta*, a drop, and is associated with the notion that some morbid matter is dropped into the joints. The disease was well known to the ancients, and its chief features have been adequately described by Greek and Roman physicians, from Hippocrates downwards.

Introductory.—Gout is a constitutional disorder characterised by paroxysmal attacks of inflammation of the joints, associated with the formation of chalk-like concretions in and about the joints. Since the discovery by Wollaston, in 1797, that these concretions contain urates, and the discovery by Garrod, half a century later, that the blood and interstitial fluids of gouty persons are surcharged with the same compounds, the opinions of pathologists have gravitated more and more steadily to the conclusion that abnormality in the destiny and disposal of uric acid is a fundamental element in any scientific conception of the gouty state, and a dominant factor in the genesis of its chief symptoms and physical signs.

Uric acid gives rise to morbid phenomena in two distinct ways; namely, in connection with gout, and in connection with urolithiasis. In both these conditions there is an aberration of uric acid; but the error is essentially different in the two cases, both as regards its site and as regards its nature. In gout the error occurs in the true interior of the body, above the limitary membrane of the secreting structures of the kidney—that is to say, in the blood and tissues—and the uric acid is precipitated in a state of combination as a urate. In urolithiasis, on the other hand, the error occurs below the limitary membrane of the kidney, in the secreted urine, and the uric acid is precipitated in the free state, on what is, strictly speaking, the exterior of the economy—that is to say, on the surface of a doubling of the external integument.

Symptoms and course of regular gout.—The first invasion of gout is usually unexpected. A middle-aged man, in the midst of apparent health, is suddenly seized in the night or early morning with pain in the ball of one of the great toes implicating the metatarso-phalangeal joint. By morning the joint is red, tense, swollen, immovable and exquisitely tender. These symptoms are accompanied by sympathetic febrile movement, scanty, high-coloured sedimentary urine, parched tongue, thirst, and anorexia. The patient is unable to put any weight on the ailing foot, and he is forced to remain in bed, or at least to keep his room. In the course of the day succeeding the onset there is usually considerable remission of the symptoms; but with the advent of evening these return in aggravated intensity, and the second night of the attack is restless,

sleepless, and marked by severe pain and rising fever. In the course of the second and third days the swelling about the joint increases, and as this goes on the tension and pain diminish, and the fever tends to subside. In a week or ten days the paroxysm exhausts itself; the tumefaction, stiffness, and tenderness pass away, and health is restored. Such is often the history of a first "fit of the gout." Not infrequently, however, the mischief is not confined to the ball of the great toe—other joints are implicated: the tarsal and metatarsal joints partake in the disturbance, and the joints of the opposite foot are involved, either at the same time or in quick succession. The paroxysm is then of longer duration, and extends to two or three weeks before final subsidence. The recovery from the first attack of gout is usually speedy and complete—the joints regain their normal size and suppleness, and the general health is perfectly re-established.

After the initial attack of acute sthenic gout, such as that above described, there is often an interval of one, two, or three years before another visitation. But the tendency to recurrence usually becomes more and more pronounced as years go by; and the gouty man at length finds himself liable to an attack once a year, or twice a year, in the spring and autumn seasons, with some approach to periodic regularity. As the disease fastens on the constitution a larger number of joints are involved; the knees become affected, the joints of the hands, and the wrists and elbows; the attacks assume a subacute character, and the condition called chronic gout is entered upon. Recovery from the paroxysms then becomes less complete, the recurrences more frequent, and the disturbance more lingering and persistent; some of the joints become permanently enlarged, stiffened, and deformed, and chalk-stones make their appearance on the knuckles and toes, and about the knees and elbows. If the disease proceed unchecked life becomes almost a continuous martyrdom, and the constitution is seriously impaired. Gout, however, is not often the direct and immediate cause of death. More frequently it kills by one of its complications—secondary inflammation, chronic Bright's disease, embolism, thrombosis, or arterial degeneration. Fortunately it is only exceptionally that gout proceeds in this inveterate fashion; more commonly, under the influence of change of habits aided by medical treatment, the downward course is arrested or mitigated; the recurrence of the attacks is diminished, and their severity and duration lessened. It is often the case that as years advance the gouty manifestations tend to decrease; and not infrequently persons, who during the middle periods of life have suffered severely from gout, attain in old age to a state of good, or at least fair health; their gout is either greatly subjugated or has entirely ceased to trouble them.

Some of the features of regular gout above sketched require more detailed consideration. The *recurrence of gouty paroxysms* exhibits great uncertainty. The disease sometimes exhausts itself in a single paroxysm; or the second attack may only occur after the lapse of decades. I knew a Yorkshire squire who had a typical seizure of gout at the age of twenty-seven;

his next attack, when I saw him, befell him in his eighty-ninth year. In the milder forms of gout it is by no means uncommon to find that the history of the disease is comprised in the incidents of two or three or half-a-dozen attacks, scattered irregularly over a long life; or perhaps compressed into the ten years of middle age. The more common rule, however, is for attacks to occur once a year or twice a year. Even in these ordinary examples breaks in the regularity of the periodicity constantly occur: either an attack comes before its time, or is postponed, or altogether omitted; or an intermediate visitation happens. In very chronic intractable cases all regularity is lost, and one attack almost runs into another; or the attacks are suspended for two or three years and then recur again as beforetime. These irregularities and vicissitudes are often quite inexplicable; in many instances they are dependent neither upon medical treatment nor upon altered dietetic habits, but are due to spontaneous changes in the constitution. They form part of the natural history of gout; and it is important to bear their existence in mind when we seek to estimate the value of therapeutic means, in order to prevent ourselves from becoming the dupes of misinterpreted sequences.

Premonitory signs.—A gouty man often has premonitions which warn him that a seizure is impending. These warning signs may prevail for many days, or even weeks before the actual breaking out of the attack. They are exceedingly diverse. The most common are digestive troubles —loss of appetite, flatulence, and acidity—with irregular action of the bowels. Very often the most prominent signs are various nervous disorders—cramps in the leg, restlessness and irritability of temper, neuralgia, pricking pain in the joints, irritating cough, unaccountable depression of spirits, persistent weariness, headache and palpitation of the heart. All these symptoms disappear at once with the onset of the arthritic attack. But it frequently happens that no warning is sounded: the seizure comes on unexpectedly, and is a disappointing surprise to the patient, who perhaps at the time was feeling unusually well, and had no presentiment whatever of the coming storm.

The *fever* which accompanies a gouty paroxysm is not usually high; it varies in degree with the severity of the local inflammation and the number of joints affected. The appearance of an inflamed gouty joint is peculiar, and suggestive of the reigning diathesis. The joint is at first tumid, tense, shining, red and very tender, and the cutaneous veins near it are enlarged. As the swelling increases the pain lessens; later the swollen parts pit on pressure, and, finally, desquamate as resolution is achieved. Suppuration does not attend on ordinary gouty inflammation; but when the larger joints and bursæ are implicated there is often copious effusion of serous fluid into their cavities.

Joints affected.—Gouty inflammation has a characteristic tendency to attack the metatarso-phalangeal articulations of the great toes. These joints rarely escape, and often they are the only joints implicated. Of 516 cases of gout collated by Scudamore the local manifestations were

confined in no less than 341 instances to one or both great toes. The next most frequently attacked joints are those of the ankle, instep, fingers, wrists, and knees. The elbow is much less frequently affected; the hips, shoulders, the articulation of the jaw, and the vertebral joints are but rarely involved. Joints which have been damaged by some previous accident or disease are more prone to take on gouty inflammation than sound joints.

Uratic deposits (chalk-stones, tophi, gouty concretions).—The occurrence of deposits of urates in the tissues of the body is the most characteristic feature of the gouty state. When a joint is the seat of gouty inflammation uratic deposits take place in the cartilages and fibrous structures of the affected articulation. Such deposits, when confined to the interior of the joints, are of course not clinically recognisable. But, although hidden from view, there is reason to believe, as will presently appear, that they are an invariable incident of an arthritic attack, and are moreover the actual excitant of the inflammatory process.

In recurrent long-continued gout these deposits are not confined to the interior of the articulations. They take place, also, in the parts outside and around the joints—in the ligaments, tendons, and subcutaneous tissue; they also invade the tendinous sheaths and synovial bursæ in the vicinity of joints, and thereby cause enlargements, thickenings, cripplings, and deformities of the implicated limbs. They then become a prominent feature of the clinical portraiture of gout, and constitute the source of the chief symptoms and physical signs of the complaint. Gouty concretions (or tophi) are essentially composed of sodium biurate disseminated, in felted masses of crystalline needles, through a matrix of connective tissue. In this way are produced the knobby, bulbous fingers and the distorted toes and knees of gouty persons. In old-standing cases gouty concretions may attain to an enormous size. Masses as large as walnuts, and even as large as small oranges, are now and then seen in the neighbourhood of the elbows and knees. When the deposits approach the surface they appear as yellowish-white masses through the skin, and occasionally they push through the cutaneous covering and form indolent ulcers over the knuckles, finger-joints, or toes, and discharge a purulent ichor containing myriads of needles of sodium biurate.

Uratic concretions are not restricted to the vicinity of joints and synovial sacs. They are found in the cutaneous and subcutaneous tissues of the palms of the hands and the soles of the feet, in the sclerotic coat of the eye, and in the superficial aponeuroses of the limbs. The most frequent seat of abarticular gouty concretions is, however, the skin of the ear. Sir A. Garrod gives the following interesting description of their formation in this situation:—"The earliest appearance presented is that of a small vesicle under the skin of the helix, as if situated between it and the fibro-cartilage. The contents of the vesicle are at first opalescent or milky, but afterwards become white and opaque, and acquire the consistence of cream. After some months the vesicle assumes the appearance of a small hard and white bead, closely resembling a pearl, and it may

remain as such for years; but occasionally the thin skin is worn off, and the bead itself becomes detached from the cartilage, leaving only a slight indication of its presence. If the vesicle is punctured in the early stage a milky fluid exudes, which presents under the microscope the appearance of a transparent liquid, in which are floating a large number of very fine crystalline needles. If the contents are examined at a later stage the crystals are found aggregated into small bundles; if the bead is solid it is difficult to separate them, as they adhere strongly together, and form a closely interlaced crystalline mass" (3).

The existence of visible uratic deposits is far from being constant even in long-standing cases of recurrent gout; but when discovered they constitute an invaluable diagnostic mark.

The number and extent of the visible uratic deposits in a gouty man present every degree of variation.

The severity of the associated symptoms is by no means proportional to the extent of the uratic deposits. Patients with numerous and enormous chalk-stones often suffer comparatively little. Other patients with few or no visible concretions suffer greatly. It would appear as if the irritation and inflammatory disturbance caused by uratic deposits depended more on their site than on their extent. Even slight deposits taking place in the interior joints may cause acute suffering; but immense deposits may accumulate outside the joints, or in detached bursæ, without provoking symptoms. The pearly concretions on the ears of the gouty rarely attract the attention of the patients.

Irregular gout.—In the intervals between the arthritic paroxysms gouty persons, especially in the earlier periods of the complaint, often enjoy perfect health, and are free from symptoms of any kind; but this is not always the case. In chronic and long-continued cases, where repeated attacks have occurred, the gouty man is well aware that his enemy is not altogether dormant even during the intervals; and he is troubled more or less with certain disturbances and symptoms which he recognises as gouty. These disturbances go by the name of irregular or suppressed gout; they are of the most varied character, and may implicate any of the functions or systems of the body. They are sometimes premonitory of an attack of regular gout, but often they come and go without reference to the occurrence of any arthritic seizure. Symptoms of a similar or kindred character may show themselves in persons who have never had any regular articular paroxysms, and who do not present any visible uratic deposits. In cases of this class the connection of the symptoms with the gouty diathesis is often not a matter of diagnostic certainty, but rather an inference from their otherwise inexplicable origin, or from the existence of a gouty family history.

Retrocedent gout. — The inflammation in a gouty joint sometimes subsides suddenly, either from some unknown cause, or as the result of exposure and chill, or of the imprudent application of cold to the joints. Such sudden subsidence of the local manifestation is liable to be followed by the development of alarming symptoms in some internal organ—the

brain, stomach, or heart. This kind of metastasis is attended with grave danger to life.

Gouty affections of the skin.—Cutaneous affections are frequently seen in the gouty, and their direct relation to the diathesis is quite undoubted. The most distinctive is eczema. The favourite seat of gouty eczema is the external ear and the parts around, namely, the face, forehead, and the back of the neck. As a rule gouty eczema is not severe, although very persistent, and is mostly confined to the parts mentioned. Sometimes, however, it becomes a grave trouble, especially in persons of advanced years, and spreads over a large part of the body. Patches of psoriasis or dry scaly eczema, fixed, circumscribed, and often unsymmetrical in position, sometimes make their appearance on the legs and elsewhere. Pruritus, either local or more generally diffused, is not uncommon, and is occasionally troublesome, particularly at night. The nails of gouty people sometimes give trouble; they become ribbed and brittle, and difficult to keep in trim order.

Gouty affections of the eye.—Various affections of the eye have been observed in gouty persons. The most common are conjunctivitis and sclerotitis. In two such cases Garrod detected uratic deposits on the sclerotic coat. Gouty iritis and glaucoma have also been described. Mr. Jonathan Hutchinson has called attention to the occurrence of hæmorrhagic retinitis in connection with gout. This condition comes on suddenly, and is always unilateral; it is probably occasioned by venous obstruction and thrombosis of the retinal vein.

Gouty affections of the nervous system.—The nervous disturbances observed in gout include recurrent headache, persistent depression of spirits, and various forms of neuralgia or neuritis. Among the latter sciatica is one of the commonest. Facial neuralgia is sometimes exceedingly troublesome; it interferes with the ingestion and mastication of food, and the patient may be obliged to have all his food finely cut up for a time, or he may even be reduced to spoon meat. In other cases the neuralgic pains affect the abdominal viscera, or shift irregularly from place to place. In rarer instances the nervous disturbances assume a more serious form and go on to delirium, insanity, or epilepsy.

The circulatory system is affected in gout in various ways. Palpitation of the heart with faltering, interrupted pulse and syncopal threatenings are not infrequent. A very peculiar paroxysmal disturbance of the circulation is now and then witnessed, in which the cardiac pulsations become extremely rapid; and a condition is produced which may be termed a "runaway heart." Paroxysms of this kind are very alarming and compel the patient for a time to lie down. Sometimes the disturbance assumes the features of angina pectoris, and constitutes a "false angina," which may be symptomatically indistinguishable from true angina; cases of this kind, which are unassociated with organic lesions of the heart, are to be carefully distinguished from cases in which true angina occurs as a complication of the gouty state. There is no direct connection between gout and pericarditis or endocarditis,—in which respect gout differs conspicuously

from rheumatism. When, however, the kidneys are involved and the gouty form of Bright's disease is developed, there is, as in other forms of granular kidney, a liability to pericarditis of very fatal tendency. Gouty persons exhibit a marked proclivity to the formation of clots in the veins (thrombosis); the seat of these is usually in the upper or lower extremities. When this tendency exists there is liability to apoplectic seizures from the formation of such clots in the cerebral veins.

The respiratory organs not infrequently feel the influence of the diathesis; and pneumonia, bronchitis, and asthmatic paroxysms are among the disorders to which gouty persons are liable.

The urine in gout.—The state of the urine in gout varies greatly according to the acute or chronic phase of the complaint, and according as the disease is or is not complicated with organic changes in the kidneys. During paroxysms of acute gout, occurring in otherwise healthy persons, the urine presents the usual features of a febrile urine: it is high-coloured, acid, and scanty; and on standing and cooling it deposits an abundant sediment of pink or brick-dust urates. If the fever run high the urine may contain a trace of albumin; but not more, nor oftener, than in equivalent pyrexia from other causes. The copious deposits of amorphous urates during acute attacks of gout have led to the belief that there is an excessive excretion of uric acid at these periods; but this is not really the case. The analyses of Sir Alfred Garrod indicate that in the early days of an attack the daily excretion of uric acid falls rather below the normal average. As the attack subsides, indeed, there is some increase, but this increase is not peculiar to gouty inflammation; it partakes rather of the nature of the critical discharges of uric acid, which are common to commencing resolution in all forms of acute inflammation.

Much more interest attaches to the state of the urine in the intervals between the gouty paroxysms than during the attack itself, when the local inflammations and the associated pyrexia mask the influence of the reigning diathesis. The most trustworthy information on this subject has been supplied by Sir A. Garrod. He divides the cases examined by him into two groups. The first group consisted of seventeen cases of chronic gout, in the majority of which there were no very urgent symptoms, but in which such sequels of the disease occurred as uratic concretions in different parts of the body, and stiffened and deformed joints. In these seventeen cases the urine was carefully analysed over a series of days. In all of them the daily discharge of uric acid was greatly below the normal average, which may be taken to be about 8 grains per day. The highest recorded daily discharge was only 5·78 grains, and that only on a single day. The average for the whole number stood under one grain per day. In several instances the total quantity of uric acid in the day's urine was so small that it could not be weighed; and sometimes the addition of an acid failed to show even a trace of uric acid in the secretion. As a rule the urine in this group of cases was copious, of low density, and paler than in the healthy state; and in more than half the cases a small amount of albumin was present in the urine. The second

group consisted of six persons who were subject to occasional attacks of gout, but who were, at the time of the examination, completely free from symptoms. In none of this group was there any albumin in the urine. The daily excretion of uric acid in all of them was found to be below the normal mean; in some it scarcely exceeded one-third or one-sixth of the usual quantity. In both these groups of cases, as well as in cases of acute gout, the daily excretion of urea did not show any appreciable deviation from the normal rate.

In some cases of chronic gout there is a pronounced tendency to *uric acid gravel.* This tendency is shown either in the deposition of free uric acid in the freshly-voided urine, or in the formation of concretions of uric acid in the precincts of the kidneys, causing attacks of renal colic followed by the passage of calculi along the ureters. These manifestations often assume a paroxysmal character, and the paroxysms of gravel appear to alternate roughly with the arthritic paroxysms. Seldom or almost never do gravelly paroxysms coincide with the arthritic paroxysms. It would at first sight appear paradoxical that uric acid gravel should occur in chronic gout, where, as we have seen, the excretion of uric acid is diminished. The explanation of the apparent contradiction lies in the fact that in the interparoxysmal periods of chronic gout the urine is, in many cases, persistently of low density and almost devoid of pigment, which condition, as I have elsewhere shown, is favourable to the deposition of free uric acid, even when the percentage of that substance in the urine is below the normal standard.

It is not to be assumed from the foregoing observations that a defective excretion of uric acid is an invariable feature of the gouty diathesis. A number of published analyses show that gouty persons may void uric acid at the full average rate, or even above that rate; and it is within the knowledge of every experienced clinical observer that men who are undoubtedly gouty, and who have gone through typical attacks of gout, may present a urine which contains its full complement of uric acid, and is in all respects perfectly normal. Such cases are not difficult to explain. In the first place, the excretion rate of uric acid is a variable one; it varies with what may be called the physiological idiosyncrasy of the individual; and an excretion rate which is equal to the general average may be a defective rate for a particular individual. Again, a gouty man is not at all times actively gouty. The diathetic tendency often remains dormant for long periods—even months and years. During these periods of abeyance the urine is in every respect perfectly normal, and shows no appreciable defect in the excretion of uric acid.

The blood in gout.—The serum of the blood in gout is impregnated with urates; that is to say, a condition which may be termed *uratæmia* prevails. This fact was first discovered by Sir A. Garrod in 1847, and has since been amply confirmed. Traces of urates exist, often at least, in normal blood, but so minute that they can only be detected by an elaborate process of analysis. In the gouty state, on the contrary, the blood is so surcharged with urates that their presence in the serum can be easily

demonstrated by a simple method, devised by Garrod, and called by him the "uric acid thread experiment." This experiment is carried out in the following manner:—A couple of drachms of the serum are placed in a watch-glass and mixed with twelve drops of strong acetic acid. One or two ultimate fibres from a piece of linen fabric are then immersed in the acidulated serum. The watch-glass is then set aside in a warm place for a day or two until the serum is evaporated almost to dryness. The watch-glass is then placed on the stage of the microscope and examined with a magnifying power of fifty or sixty diameters. If the blood contain urates in excess, minute rhombic crystals of uric acid will be found sprinkled upon the submerged threads; if the blood be normal, no such crystals will be discoverable. By the same method Garrod discovered that in gouty persons the serum effused by the application of a blister is similarly charged with urates. In making this observation he found that it was necessary to raise the blister on a portion of the skin not involved in the gouty inflammation, for blister serum obtained from the surface of an inflamed gouty joint contained no urates; and he inferred therefrom that uric acid was destroyed in the process of gouty inflammation. The same observer states that during the period of convalescence from acute gout there is a marked diminution of urates in the blood. In the long intervals which, in the earlier periods of the disease, often elapse between the attacks, no appreciable amount of urates is discoverable in the blood; but in inveterate cases the blood, even in the intervals between the exacerbations, was always found surcharged with urates.

Gout is, however, not the only morbid condition in which urates are in excess in the blood. Such excess has been found also in cases of leukæmia, pneumonia, anæmia, and Bright's disease.

Morbid anatomy.—The anatomical changes which are characteristic of gout depend essentially on the uratic deposits which form in various parts of the body, especially in and about the joints; and on the inflammatory and degenerative processes which immediately or remotely follow thereupon.

When a gouty joint is dissected after death the articular cartilages are seen to be encrusted with a white mortar-like material which, on analysis, is found to be composed of sodium biurate. This substance is scattered in specks, streaks, or patches on the cartilages. In some instances the deposit is strictly confined to the articular cartilages; in others it extends to all the structures of the joint—to the ligaments, fascias, tendons, and the synovial membranes; and the synovial fluid itself is charged with specks and crystals of the same substance. When the joints are enlarged and deformed the deposit infiltrates the tissues outside and around the articulations—namely, the subcutaneous connective tissue and the substance of the skin—penetrating towards the surface and forming chalk-stones and tophi. The synovia is generally found to be thick and scanty; but sometimes in the case of the larger joints, such as the knees, there is a considerable effusion of fluid into the cavity of the articulation.

Remote secondary changes are often discovered in old gouty joints, erosions of the articulating surfaces and thickenings of the ends of the bones. These latter changes, however, are not peculiar to gouty joints, but are identical in nature and character with those which are produced by repeated and chronic inflammations of the joints from rheumatism or other cause.

The commonest and indeed often the only seats of uratic deposits found after death are the articular cartilages, and these have been the most carefully studied. On close examination it is perceived that the deposit is not really on the surface of the cartilage, as at first sight it appears to be, but is situated interstitially in its substance. This may be demonstrated by cutting a thin vertical section through the cartilage at the site of the deposit, and examining the section under the microscope. It is then seen that the uratic matter is infiltrated through the matrix of the cartilage beneath the free surface, and that it is composed of felted masses of acicular crystals. The deposit penetrates only to a small depth below the surface, usually to less than one-third or one-half of the entire thickness of the cartilage. It is further seen that the deposit is densest close under the articulating surface, and that it grows thinner and thinner towards the deeper parts. The deepest layers near the bone, as well as the bone itself, are entirely free from deposit. It is where the deposit is thinnest that its crystalline structure is most clearly discerned.

When thin sections of a gouty cartilage are digested for some hours in warm water, the white matter is entirely dissolved out, and the tissue to the naked eye reassumes its normal appearance. But when sections thus treated are examined under the microscope, it is seen that in those parts where the deposit was thick and dense the matrix of the cartilage is profoundly altered, and displays an opaque granular appearance, or may be even eroded; but that in spots where the deposit was thin and sparse the matrix shows no change from its normal aspect. These appearances, which have been verified over and over again, prove that the uratic deposits in gouty joints are thrown down in previously healthy cartilage—that the morbid changes found therein are not primary, but secondary, and are due to the pressure and irritation of the deposited matter. Moreover, the fact that the deposit is densest near the articular surface of the cartilage, and thins away towards the deeper layers, indicates that the uratic matter had its source in the synovial fluid bathing the cartilage, and was precipitated from it.

The causal relation between uratic deposits and gouty inflammation of the joints has been minutely traced by Sir A. Garrod. He made an extended series of observations on the post-mortem appearance of the joints of gouty persons whose previous morbid history had been carefully ascertained; and he found that those joints which had passed through an attack of gouty inflammation at some previous period of life always displayed the accusing uratic deposits at the autopsy; whereas those joints which had not been so attacked were quite free from such deposits. On

the ground of these observations Garrod formulated the proposition that "*gouty inflammation is invariably attended with deposition of urate of soda.*" This proposition, thus guardedly worded, is probably universally true; but it does not appear to be universally true that a joint, which at some previous epoch of life has undergone an attack of gouty inflammation, will invariably be found after death to exhibit uratic deposits. Sir Dyce Duckworth records the case of a man under his care with chronic phthisis, who had had two attacks of gout in the right great toe-joint; at the autopsy neither toe-joint contained a speck of uratic deposit. This case, however, does not prove that uratic deposits had not taken place during the gouty attacks; it only proves that such deposits are not always permanent, and that, under certain conditions, they may be removed by re-solution. Nor can it be said that the converse of Garrod's proposition is invariably true, namely, that a joint which exhibits uratic deposits after death must, in every case, have undergone an inflammatory gouty attack during life. Some observations bearing on this point have been recently published by Dr. F. Levison of Copenhagen, who gives the details of the autopsies of twelve cases of chronic Bright's disease with granular contracting kidneys, in all of which uratic deposits, mostly very slight, were discovered in one or more joints. The previous clinical history of the larger number of these cases was either defective or altogether wanting; but, with regard to four cases in which the previous history was ascertained, no evidence was elicited that any inflammatory attacks in the implicated joints had occurred during life (4). The absence of inflammatory attacks in these cases is probably to be explained partly by the scantiness of the deposit, and partly by its slow formation.

The relative frequency with which the several joints are affected with uratic deposits corresponds closely with the relative frequency with which they are subject to gouty inflammation as clinically observed. This broad conclusion is clearly brought out in the records of eighty autopsies of gouty subjects made by Dr. Norman Moore, and published in St. Bartholomew's Hospital Reports for 1887. The most commonly affected joints are the metatarso-phalangeal joints of the great toes; and not infrequently these are the only joints affected: then follow the joints of the insteps, ankles, knees, hands, and wrists. The elbows, shoulders, and hips are more rarely affected. Deposits may be present in nearly all the joints of the lower extremities, and yet be entirely absent from those of the upper extremities. Among the rarest sites of uratic deposits may be mentioned the articulations of the jaws and larynx and the sterno-clavicular joints.

As already mentioned, uratic deposits occur in other parts than the joints and their neighbourhoods. The most common sites of these *abarticular deposits* are the rim of the ear, and the tendons and aponeuroses in various parts of the body. Such deposits are also seen not infrequently in the skin of the palms and soles, on the eyelids, nose, and other parts of the face; also, more rarely, on the vocal cords, the

cranial and spinal dura mater, the pia mater, the sclerotic coat of the eye, the fibrous sheaths of the nerve-trunks, and the aortic valves.

The kidneys in gout.—It has been long recognised that there are intimate relations between gout and renal disease. The form of kidney disease which is especially associated with gout is that which is characterised by granular degeneration and atrophy of the organs. Hence this form of renal disease is sometimes spoken of as the "gouty kidney," or "the gouty form of Bright's disease." The relations between articular gout and renal disease are both complicated and inconstant. In many cases the renal affection appears distinctly as a sequence of the arthritic. In other cases signs of renal disease precede the arthritic manifestations; or again the two conditions may arise simultaneously. Sometimes, as before pointed out, the necropsy of subjects affected with granular contracting kidneys discloses the existence of unsuspected uratic deposits in the joints, where the clinical history had indicated no antecedent inflammatory arthritic attacks. It may be said, generally, that the coincidence of articular gout with renal disease is more common among the poor and in hospital patients than among the easier classes. In cases of saturnine gout implication of the kidneys is almost if not quite invariable. On the other hand, in private practice, and among well-to-do patients, it is quite common to see articular gout, even of a chronic and inveterate character, run its entire course without any accompanying signs of structural disease of the kidneys.

When gouty kidneys are cut open white deposits of uratic matter are seen on the cut surfaces. In the cortical substance the deposit occurs as specks scattered irregularly through the tissue; but in the pyramidal portion the matter is in streaks running in the direction of the tubuli. In both cases the deposit is situated in the intertubular substance; and, when examined under the microscope, is seen to consist of acicular crystals exactly resembling those found in gouty joints.

Pathology and chemistry of gout.—Gout may be regarded as the expression of a peculiar diathesis, or constitutional bias, which is either inherited from a gouty ancestry, or is acquired during life through the operation of extrinsic causes. The distinctive features of the diathesis are a tendency to (*a*) the accumulation of urates in the blood, and (*b*) to the deposition of urates in the tissues.

These two conditions, to which the names *uratæmia* and *uratosis* may be respectively applied, are by no means of identical pathological valency. They differ from each other in several important respects. In uratæmia the urates circulate in a state of solution in the blood and lymph—in uratosis the urates are deposited in the solid form in the substance of the tissues. In the former condition the urates can only act injuriously, if they so act at all, after the manner of a chemical poison; in the latter condition the deposited crystals must act more or less as a mechanical irritant. Uratæmia, I repeat, is not confined to gout; it occurs also in anæmia, leukæmia, pneumonia, and some other conditions, which cannot by any stretch of definition be included in the term gout.

Uratosis, on the other hand, is absolutely confined to the gouty state, and constitutes its pathognomonic mark. Uratosis cannot occur, so far as is known, without coexisting uratæmia; but it is certainly true that uratæmia may exist for long periods without provoking the occurrence of uratosis. From these differences it may be inferred that the conditions of origin of uratæmia and of uratosis are not quite the same; and that in uratosis there are some additional factors in operation beside and beyond those which give rise to uratæmia. It is evident, moreover, that there is a closer relation between gout and uratosis than between gout and uratæmia; and that the conditions which lead to uratosis have a more immediate bearing on the pathology of gout than the conditions which lead to uratæmia.

Chemistry of Uratosis.—It has already been shown that gouty deposits are essentially composed of crystals of sodium biurate. It has also been shown to be extremely probable, if not certain, that the precipitation of these crystals in the joints is the actual excitant of the arthritic attacks in gout. A knowledge of the chemical relations of sodium biurate is therefore indispensable to a clear understanding of the gouty process. It is of interest to know how this compound arises in the bodily fluids, what are its relations of solubility in diverse media—especially in the blood, lymph and synovia — and what are the factors which tend to determine its precipitation in the tissues, or tend to prevent its precipitation.

It is to be remembered that uric acid does not exist in the blood and lymph in the free state, but in a state of combination—as a urate. When, therefore, uric acid is spoken of as circulating in the bodily fluids the expression is not quite exact; what really circulates is not uric acid, but a urate. There are three classes of urates which differ radically from each other both in their chemical properties and in their conditions of origin, namely, *neutral urates*, *biurates*, and *quadriurates*. The neutral urates ($M_2\ \bar{u}$) are only formed when uric acid is brought into contact with alkalies in the caustic state; and, as caustic alkalies never exist in the animal body, the neutral urates can never intervene in the physiological or pathological history of uric acid. The biurates ($MH\ \bar{u}$), in the form of the sodium biurate, exist, as has been seen, in gouty concretions. The quadriurates ($H_2\ \bar{u}$, $MH\ \bar{u}$) constitute the form, and the sole form, in which uric acid subsists in normal urine. The amorphous urate sediment of human urine is thus composed, and consists of a mixture of the quadriurates of potassium, sodium, and ammonium in varying proportions.[1]

It may be further said that the normal or physiological status of uric acid in the body is that of a quadriurate, and that any departure from this status leads to pathological consequences. In urolithiasis the departure is towards a breaking-up of the unstable quadriurate, and a

[1] For a fuller account of the urates and the chemistry of gout, the reader is referred to the author's Croonian Lectures for 1892 *On the Chemistry of Uric-Acid Gravel, and Gout*, published by Smith, Elder, and Company.

setting free of its uric acid, thereby leading to the formation of urinary gravel and calculus. In gout the departure is in the converse direction —towards the transformation of the quadriurate into the more stable biurate, and the precipitation of the latter in the tissues.

Seeing that sodium biurate is the essential component of gouty concretions, the solvent relations of this substance are of great interest in the study of the pathology of gout. The best solvent for sodium biurate is pure water, which, at blood-heat, dissolves it in the proportion of about 1 part in 1000; but if any salt of sodium be added to the water its solvent power on the biurate is greatly reduced. Water containing 0·2 per cent of the bicarbonate or chloride of sodium only dissolves about 1 part in 3000; water containing 0·5 per cent only dissolves about 1 part in 10,000; and in water containing 0·7 per cent of sodium salts the biurate is almost insoluble. All the sodium salts act alike; the carbonate and phosphate, which have an alkaline reaction, exercise just as much deterrent effect as the chloride and sulphate, which have a neutral reaction. Now the serum of the blood, and its derivatives, lymph and synovia, are very rich in sodium salts,—they contain about 0·5 per cent of sodium chloride, and about 0·2 per cent of sodium bicarbonate, making with the small amount of sodium phosphate a total of about 0·73 per cent of sodium salts; whereas the other salts of the serum—potassium, calcium, and magnesium salts—only amount to 0·11 per cent all put together. It was ascertained by direct experiment that the behaviour of uric acid and the urates with blood-serum and its derivatives depends entirely on the saline ingredients which they contain, and is quite independent of their albuminous constituents. When serum was deprived of its salts by dialysis it reacted with uric acid and the urates like simple water. It was not, therefore, surprising to find that sodium biurate is almost insoluble in serum and synovia. From a number of comparative experiments it was estimated that these fluids could not hold in solution more than about 1 part in 10,000 of crystalline sodium biurate.

Results of a corresponding character were obtained with actual uratic deposits, as shown in the following experiments:—A metatarsal bone from the body of a gouty man, encrusted on its articulating surfaces with uratic matter, was suspended in a phial containing six ounces of blood-serum of the pig, a few drops of chloroform being added to prevent decomposition. The phial was placed in the warm chamber, at blood-heat, for a fortnight, and afterwards kept on a shelf in my sitting-room. The serum was renewed three times in the course of the experiment. No appreciable change in the deposit was observed for a very long time. Even after the lapse of eight months the encrusted matter did not appear diminished. Nevertheless slow solution was taking place. At the end of twelve months a diminution was visible, and in fifteen months the deposit was entirely dissolved out. In strong contrast with this was the behaviour with water. A second metatarsal bone from the same subject, and similarly encrusted, was suspended in six ounces of distilled

water, and treated in the same way. The deposit was entirely dissolved out in four days. The cause of this immense difference was due to the large quantity of sodium salts in the serum.

Behaviour of uric acid and the quadriurates with blood-serum and synovia.—An examination of the reaction of free uric acid with blood-serum and synovia is a necessary part of the study of the chemistry of gout. It is only by knowledge thus gained that we can hope to elucidate the mode in which sodium biurate originates in the body, and to learn something of the conditions which control its precipitation in the gouty system.

When uric acid is digested, at the temperature of the body, with serum or synovia it passes freely into solution in combination with a base. Such a solution (when fully saturated) is found to contain as much as 1 part in 500 of uric acid. The chemical and solvent power of serum and synovia on uric acid depends entirely on the sodium carbonate which they contain, which imparts to them their alkaline reaction—and has no relation to the sodium chloride and other neutral salts contained in them. The combination which is formed under these circumstances is always in the first instance a quadriurate, and not a biurate. The quadriurate is, however, an unstable salt; and, in solutions of the alkaline carbonates such as the serum of the blood, lymph, and synovia, it slowly takes up an additional atom of base, and is thereby gradually converted into a biurate. The biurate of sodium thus formed finds itself in a medium rich in sodium salts, and one wherein, as we have seen, it is almost insoluble; it is, consequently, after a time thrown out of solution and precipitated in the crystalline form.

In order to bring this remarkable succession of events more clearly before the reader's mind the particulars of three experiments may be related.

Experiment 1.—Fresh serum of pig's blood was treated with uric acid in excess in a 4-oz. phial, tightly corked, and chloroformed to prevent decomposition. The phial was gently turned upside down a few times at first, but was not subsequently disturbed; it was then placed in the warm chamber. The serum soon cleared, the undissolved uric acid fell to the bottom, and the supernatant serum became transparent. For about twenty-four hours no change occurred, but in the course of the second day stars of sodium biurate were detected amid the deposit, and during the third day an abundant precipitation took place of stars and needles of biurate, exactly resembling those found in gouty concretions. On the fourth day the process of precipitation was nearly complete, and the supernatant serum was found comparatively free from uric acid.

Experiment 2.—Blood-serum of the horse was digested at 100° F. with excess of uric acid for fifteen minutes with constant agitation. The serum (now highly charged with dissolved quadriurate) was then filtered and placed in a corked phial in the warm chamber. In about twelve hours the serum, previously clear, began to lose transparency, and fine needles of biurate were detected in it with the microscope. On the next day copious precipitation took place. On the fourth day the process

seemed to be complete, and the supernatant serum was found to contain only traces of uric acid.

Experiment 3.—Synovia obtained from the hip-joints of an ox was shaken up with excess of uric acid for about half an hour, and then left at rest. In six hours the undissolved uric acid had subsided. The supernatant fluid was then carefully decanted into a 2-oz. phial and placed in the warm chamber. For two days it remained quite unaltered; but on the third day precipitation of biurate began, and continued copiously during the fourth day; on the fifth day precipitation was apparently completed, and the supernatant liquor was found almost free from uric acid.

It was impossible not to be struck with a certain rough resemblance between the results observed in these experiments and the phenomena of the gouty paroxysm. In the gouty subject it is assumed that the blood becomes more and more impregnated with urates until, after a certain period of incubation has been accomplished, sudden precipitation of sodium biurate takes place in and about the joints, and the "fit of the gout" is declared. Then follows a process of recovery, with restoration of the blood to a purer state—that is, with a lessened impregnation with urates. In the artificial counterfeit we observe a similar succession of events: first, impregnation of the medium with sodium quadriurate; secondly, a period of incubation or maturation, during which the quadriurate passes gradually into biurate; thirdly, somewhat sudden precipitation of sodium biurate in the crystalline form; and, lastly, restoration of the medium to comparative purity.

There is a point of some importance in connection with the chemistry of the biurates which may be here mentioned. The biurates are capable of existing under two distinct forms or modifications, namely, the ordinary anhydrous crystalline form seen in gouty concretions, and a hydrated gelatinous modification. The hydrated modification is much more soluble in serum and other media than the crystalline form. The hydrated condition is essentially unstable, and it tends to lapse by a spontaneous change, into the crystalline form.

The facts above elicited respecting the chemical relations of uric acid and the urates enable us to obtain a coherent view of the history of uric acid in the living body. In normal urine uric acid always exists as a quadriurate; in animals, such as birds and serpents, which eliminate their nitrogen as uric acid, the urinary excretion is composed entirely of the same combination. It is, moreover, demonstrable that in media containing alkaline carbonates—such as the serum of the blood and its derivatives, lymph and synovia—uric acid passes into solution in the first instance as a quadriurate. From these considerations it may be inferred that in the normal state uric acid is primarily taken up in the system as a quadriurate; that it circulates in the blood as a quadriurate; and that it is finally voided with the urine as a quadriurate. In perfect health the elimination of the quadriurate proceeds with sufficient speed and completeness to prevent any undue detention or any accumulation of it

in the blood. But in the gouty state this tranquil process is interrupted, either from defective action of the kidneys or from excessive introduction of urates into the circulation, and the quadriurate lingers unduly in the blood and accumulates therein. The detained quadriurate, circulating in a medium which is rich in sodium carbonate, gradually takes up an additional atom of base, and is thereby transformed into biurate. The biurate thus produced exists at first in the hydrated or gelatinous modification; but with the lapse of time and increasing accumulation it passes on into the almost insoluble anhydrous or crystalline modification; and then precipitation of it becomes imminent, or actually takes place.

The conditions which accelerate or retard the processes which culminate in the precipitation of sodium biurate.—Assuming a real analogy to exist between the processes which go on in serum artificially impregnated with uric acid, and the processes which go on in the blood of a gouty patient, and culminate in the deposition of uratic concretions, it is a matter of interest, as bearing on the pathology and treatment of gout, to investigate the conditions which, in the artificial parallel, accelerate or retard these processes.

As already explained, these processes consist of three distinct chemical changes. First, the quadriurate originally formed is converted into hydrated biurate; next, the hydrated biurate is changed into anhydrous biurate; and, finally, this anhydrous biurate is precipitated in the crystalline form. For the present purpose it will be more convenient to consider these changes as one continuous process, and for the sake of brevity and ease of expression this process may be designated as *maturation.*

The investigation embraced a study of the effects of temperature, percentage of uric acid in solution, and the addition of various saline and other substances to the maturating medium.

(*a*) Temperature.—It was found invariably that maturation was more quickly accomplished in the warm chamber at 100° F. than at the temperature of the room, but the ultimate result was exactly the same in both cases. For example, serum charged with 1 part of uric acid in 600 began to precipitate in the warm chamber in four hours, and precipitated copiously in six hours. A duplicate specimen kept at the temperature of the room (65° F.) began to precipitate in eight hours, and did not precipitate copiously for sixteen hours.

(*b*) Quantity of Uric Acid in Solution.—It was found that no factor exercised so great and decisive an influence on the speed of maturation and the advent of precipitation as the proportion of uric acid in solution. The copiousness of the precipitation was likewise, of course, affected by the same factor. The following experiment with blood-serum, the results of which are arranged in a tabular form, illustrates these points in a striking manner:—

TABLE showing the Influence of Percentage of Uric Acid in the Medium on the Speed of Maturation and the time of Advent of Precipitation.

Quantity of Uric Acid contained in the Serum.	Time of Precipitation of Sodium Biurate.
1 in 1000 . . .	Precipitation began in six hours, copious precipitation in fourteen hours.
1 in 2000 . . .	Precipitation began in thirty-three hours, copious precipitation in three days.
1 in 3000 . . .	Slight precipitation began in three days, which became a little more copious in twelve days.
1 in 4000 . . .	A few needles of biurate were detected on the sixth day; more needles and a few tufts in twelve days.
1 in 5000 . . .	A few short needles were detected on the thirteenth day. In thirty days the needles were somewhat more numerous.
1 in 6000 . . .	No needles were discoverable in fourteen days; a few were detected in forty days.
1 in 8000 . . .	No needles could be detected after a lapse of forty days.

Assuming that the inflammatory arthritic attacks in gout are directly due to copious and sudden precipitation of crystalline stars and needles of sodic biurate in the cartilages and fibrous structures of the joints, the evidence before me indicates that such copious and sudden precipitation can only take place when the fluids bathing these structures are impregnated with uric acid in at least the proportion of 1 part in 2500. Below this point the precipitation occurs slowly and scantily, and only in the form of short scattered needles. When the proportion of uric acid dissolved in the serum was only 1 part in 5000 the deposited needles of biurate were mostly about as long as the diameter of a red blood disc, some were twice this length, and a few three times this length; all were of extreme tenuity. It is quite conceivable that this slighter precipitation in the tissues of short scattered needles might account for certain irritations in the various organs, such as characterise irregular or larval gout, but it could scarcely engender frank inflammatory attacks. It is further conceivable that the presence in the blood of such scattered needles might constitute foci around which clotting might take place, and that the thrombosis not infrequently observed in gouty cases might be thus accounted for.

The impregnation of the blood in gouty persons with uric acid to the extent of these lesser degrees is within the range of observed actualities. Sir Alfred Garrod, by quantitative analysis, obtained from the blood-serum of one of his patients uric acid to the amount of 1 part in 5714; and he remarks that the quantity thus recoverable from the blood is probably much under the actual amount, as considerable loss may occur from unavoidable causes.

These considerations lead to the suggestion that a microscopical examination of the blood in gouty persons might sometimes reveal the existence of needles of biurate in that fluid. I tested this point in ten cases of chronic gout by examining a drop of blood drawn from the finger, but I failed to obtain positive results.

(*c*) Influence of Saline Substances.—The effect of saline substances on the maturating process was tested by adding small quantities of various salts to serum impregnated with uric acid, and observing whether these additions accelerated or retarded precipitation. The following summary indicates the conclusions deduced from the experiments :—

The addition of sodium salts to the maturating medium invariably hastened the precipitation. An idea of the degree of acceleration may be gathered from the following examples :—Serum impregnated with uric acid to the extent of 1 part per 1000 began to precipitate in seven hours, and precipitated copiously in sixteen hours. A parallel experiment, in which 0·2 per cent of sodium chloride had been added to the serum, began to precipitate in five hours, and precipitated copiously in twelve hours. Another sample of serum was impregnated with uric acid to the extent of 1 part in 2000. This began to deposit crystals in thirty hours, and deposited freely in ninety-six hours. In a parallel experiment, in which 0·2 per cent of sodium bicarbonate had been added to the medium, precipitation began in twenty hours, and free precipitation took place in forty hours. The alkaline reacting salts—the carbonate and phosphate—had exactly the same effect as the chloride and sulphate, which are neutral in reaction.

The addition of potassium salts sensibly retarded precipitation, but did not appreciably diminish the eventual amount of it. Here, again, the carbonate and phosphate, which are alkaline, produced just the same effects as the chloride, iodide, and bromide, which are neutral. Both with potassium and sodium salts the results were entirely dominated by the nature and quantity of the bases added, and had no reference to the acidulous radicle with which the bases were combined.

The addition of calcium and magnesium salts appeared to delay precipitation, but their action in this respect was quite insignificant or even doubtful. The salts of lithium had not the slightest influence either way. Piperazine, whether in the free state or as chloride, was not found to exercise any influence on the advent of precipitation.

Topography of uratic deposits.—The topographical distribution of uratic deposits through the various organs and tissues of the body exhibits certain well-marked characteristics. These deposits are found almost exclusively in structures belonging to the connective tissue class—in cartilages, ligaments, tendons, and other fibrous structures, and in the cutaneous and subcutaneous connective tissues. On the other hand, uratic deposits are conspicuously absent from the muscular tissue, and from the substance of the brain, liver, spleen, and lungs. The tissues which are liable to uratic precipitations are, however, not equally so in the different parts of the body. The cartilages, ligaments, and tendons

in and about the joints, which are bathed with synovia, are much more prone to these deposits than are cartilages and fibrous structures situated at a distance from joints, and which are not bathed with synovial fluid. It is further to be noted that uratic deposits favour the more superficial and cooler parts of the body, especially the upper and lower extremities, and are more rare in the deeper and warmer interior parts of the trunk. It may be inferred from these particulars that the influences which co-operate to determine the site of uratic precipitations are of several and quite different kinds. I do not propose to discuss this subject comprehensively, but to refer to two points only which seem capable of chemical or physical elucidation. These are (*a*) the influence of the proportion of sodium salts in the several organs and tissues, and (*b*) the influence of synovia.

(*a*) Influence of the proportion of sodium salts.—We have seen that the dominant factors in uratic precipitation, as studied in the laboratory, are the proportion of urates and the proportion of sodium salts contained in the medium.[1] The highest tendency to precipitation is reached when there is a concurrence of these two factors in maximum intensity. A medium may be rich in urates, but if it be at the same time poor in sodium salts, its tendency to precipitation is feeble, and *vice versa*. This fact has a direct bearing on the topography of uratic deposits. For if we suppose the system of a gouty man, on the eve of an outbreak, to be throughout equally impregnated with urates, it is obvious, from the experimental evidence before adduced, that uratic precipitation would take place earliest and most copiously in those parts which were richest in sodium salts, and take place latest, or not at all, in those parts which were poorest in sodium salts. Let us now examine the distribution of sodium salts in the body, and seek to ascertain if there be any correspondence between the liability to uratic deposits in the several tissues and organs and the proportion of sodium salts normally contained in them. In the subjoined table I have arranged the results of analyses on this point. The materials available for comparison are not so full and precise as could be desired, but their general significance is, I think, quite unmistakable. In order to render the comparison more complete and instructive I have included in the table not only the results with respect to the solid organs and tissues, but also those with respect to blood-serum and its derivatives—lymph and synovia.

TABLE showing the Percentage of Sodium Salts in the several Fluids, Tissues, and Organs of the Body.

	Sodium Salts per cent.		Sodium Salts per cent.
Blood-serum	0·70	Blood corpuscles	0·20
Lymph	0·70	Brain	0·20
Synovia	0·80	Muscle	0·08
Cartilage	0·90	Spleen	0·04
Fibrous tissue	0 70	Liver	0·02

[1] The proportion of calcium and magnesium salts is always too small to have any appreciable influence on the occurrence of uratic precipitation.

An inspection of the table shows that the tissues which are liable to uratic deposits are very much richer in sodium salts than the tissues and organs which are not thus liable. The remarkable immunity from uratic deposits enjoyed by the muscular tissue and by the brain, liver and spleen may be inferred to be due, for the greater part at least, to their poverty in sodium salts. It has before been shown that the solvent power of a medium for sodium biurate increases in correspondence with its lessening proportion of sodium salts. Brain has only about one-fourth the percentage of these salts as compared with cartilage and fibrous tissue, and muscle only one-tenth. This difference signifies (roughly) that brain has four times more power, and muscle ten times more power, of dissolving sodium biurate than cartilage and fibrous tissue, and therefore, respectively, four and ten times more power of resisting its precipitation in their substance. On this ground it might probably be truthfully said that brain, muscle, liver, and spleen could not become the sites of uratic deposits until the fibrous and cartilaginous tissues had been exhausted in this respect. It is true that the immune tissues and organs have a quicker circulation than cartilages and tendons, and this doubtless contributes importantly to the difference; but it does not fully account for it, otherwise we should expect to find that the skin, which is abundantly supplied with blood-vessels, would share this immunity. The prepotency of the cartilages and fibrous tissues to induce uratic precipitation in their substance must obviously operate in a conservative direction, and serve to protect the more vital organs of the gouty from similar precipitations, where they would produce more deadly effects.

(*b*) Influence of synovia.—The connection between synovia and gouty deposits is evidently very close and special. Synovial fluid itself has been repeatedly found heavily laden with crystals of sodium biurate. In the great majority of the less severe cases of gout uratic deposits are exclusively confined to those cartilages, ligaments, and tendons which are in actual contact with synovial sacs or synovial sheaths. With regard to the articular cartilages it may be demonstrated, I think, that the uratic precipitation actually takes place from the synovial fluid, and is not self-originating in the cartilaginous substance. Vertical sections of gouty cartilages are very convincing on this point; if such sections are examined under the microscope it is seen that the deposit hugs the free surface of the cartilage, and that it becomes progressively sparser and sparser towards the deeper layers—the central and deepest parts being often quite free from deposit.

This mode of distribution, moreover, implies that the process of deposition, so far as concerns the cartilage, is a purely passive and physical one, and in nowise active and vital in its initiation. We may suppose that the urate dissolved in the synovia penetrates by liquid diffusion into the superficial layers of the underlying cartilage, and that when the critical moment arrives precipitation takes place simultaneously in the synovia and in the cartilage. On this view the granular and erosive changes sometimes found in gouty cartilages are entirely secondary, and are due

to the reaction of the tissue against the presence of a foreign body lodged in its substance. It is of some interest to know that this process can be artificially imitated, and that a counterfeit gouty cartilage can be produced in the articulation of a dead animal. Samples of such counterfeits were procured in the following manner:—Tarsal bones of a pig were suspended in phials charged with a saturated solution of sodium biurate made in hot water and then cooled. The phials were chloroformed and corked, and then set aside in the warm chamber, or at a temperature of the room. Reprecipitation of the biurate took place in two or three days. If the bones were now examined, the articulating ends were found to be encrusted with a chalky matter, which could not be wiped off with a towel nor removed with a nail-brush. They presented an exquisite imitation of the plastered appearance of a gouty cartilage. If vertical sections of such cartilages (previously hardened in absolute alcohol) were made, and examined with the microscope, the deposit was seen to be situated in the substance of the tissue, close beneath the synovial surface of the cartilage, and could be identified as consisting of a dense felt of fine needles of biurate. The deeper layers of the cartilage were not affected.

It may be inferred that the uratic deposits which occur in the ligamentous and tendinous structures of gouty joints are produced in the same way as those found in the articular cartilages; that the precipitations take place, chiefly at least, from the synovial fluid with which they are bathed, and that they are not primarily due to morbid changes in the structure of the tissues.

Two questions may now be asked:—First, why does gouty precipitation take place preferentially in synovia rather than in its cognates, the serum of the blood, and lymph? And, second, why do the joints differ so much from each other in their liability to attack? I will discuss the two questions together, as the arguments often dovetail into each other. It may be that there is, as Sir A. Garrod suggests, some special attraction in the joints for uric acid. But there are undoubtedly other factors which come into play. Synovia is a comparatively motionless fluid, while serum and lymph are in ceaseless motion. And as a still pool crystallises into ice sooner than a running brook, so likewise—supposing serum, lymph, and synovia to be equally impregnated with urates and sodium salts—the tranquillity prevailing within the synovial sacs would give to synovia a priority in uratic precipitation over the restless blood-serum and lymph. Then, as regards the varying liability of the different joints to gouty attacks, it may be pointed out that the synovial pouches and sheaths are shut sacs, standing apart from each other, and isolated from the general panmixia of the circulation. It might consequently be expected that the synovial fluids in different joints should present differences in the relative proportions of their constituents; indeed, the fact is observed. Some are certainly more concentrated than others, and we can easily believe that they are not quite identical in the degree of their impregnation with urates and with sodium salts. In this way the several

joints might come to vary considerably in their liability to uratic precipitation.

Frerichs has contributed some interesting particulars on synovia in animals. His observations indicate that synovia varies both in quantity and quality under different modes of life. He found that stall-fed horses and oxen, leading an idle existence, had twice as much synovia in their joints as similar animals roaming in the meadows or doing work. Moreover, the composition of the fluid varied in the contrasted conditions. In the idle animals the synovia was more watery, and contained less albuminoid matters, but—and this is significant—a larger proportion of mineral salts, which consist almost entirely of sodium salts (2). We might conjecture from this observation that, if horses and oxen were liable to uratic precipitations, the idle stall-fed animals would be more subject to such deposits than the same animals leading a more active life. Perhaps we may discern herein one reason why men who lead a sedentary life are more subject to gouty deposits than men who take active exercise.

Interpretation of the appearances found at the necropsy of gouty subjects. Re-solution of gouty deposits.—Before leaving the subject of the topographical distribution of gouty deposits, a word may be said on the need of caution in interpreting the appearances, both positive and negative, observed at the necropsy of gouty subjects. The incidents of the gouty diathesis, especially in the earlier periods, pursue a markedly interrupted course. Long intervals of months or years often elapse between the arthritic outbreaks. During these intervals the blood of the gouty man recovers its purity more or less completely, and approximates in its contents of uric acid to the blood of a healthy person. The solvent relation of the bodily fluids to the material of gouty deposits is simply a question of saturation or subsaturation. If the lymph or synovia at a certain spot become saturated, or rather supersaturated, with sodium biurate, precipitation of that substance will inevitably take place into the contiguous fibrous tissues; and if, after such an event, the lymph or synovia recover its purity and become approximately free from biurate, as in due course usually comes about, a process of slow re-solution will of necessity set in. For, as has been before demonstrated, sodium biurate, although very sparingly soluble, is not absolutely insoluble in these media. The rate and amount of re-solution of uratic deposits must necessarily vary greatly in different cases—according to the degree of subsaturation attained by the bodily fluids, the massiveness and penetrability of the deposits, and the length of time during which the favourable conditions endure.

If regard be had to the, often, long survivorship of gouty persons, and the interrupted course of the arthritic incidents, it seems highly probable, in the lapse of a long life, that deposition and re-solution of uratic matter may take place once and again in a gouty joint. There are undoubted instances, as has been already pointed out, where no uratic deposits have been found after death in joints which, at some previous period,

had undergone definite gouty attacks. The presumption in such a case is, not that deposits never existed, but that in the intervals of amendment they had been redissolved. Uratic concretions in the pinna of the ear have sometimes been actually observed to come and go, and come again. There is no reason why the same thing should not occur within the joints; and such vanished concretions might, or might not, leave behind them permanent changes in the cartilages and bony structures as tell-tale evidence of their former presence. On the other hand, the discovery after death of uratic deposits in a joint is not always to be regarded as a certain proof that the joint had passed through an inflammatory gouty attack. The observations of Moxon and Fagge seem to warrant this conclusion; and they reasonably suggest that when the precipitation of the urates takes place slowly and by degrees, there may be no accompanying inflammatory outbreak to mark the event (1). The cases of Dr. Levison, before cited, also support this conclusion (see *ante* p. 161).

The experimental evidence before adduced indicates that when lymph or synovia is impregnated with sodium biurate to or above 1 part in 6000, the medium is supersaturated, and precipitation either actually occurs or is impending. On the other hand, when these fluids contain less biurate than 1 part in 10,000, the medium is undersaturated, and there resides in it a certain power of redissolving uratic deposits; the nearer the medium is to freedom from urates, the higher, of course, rises this solvent power. It may, further, be inferred that the solvent action will be most effective in the case of deposits situated in textures like the fibrous tissues, which have a comparatively free lymph flow; and, conversely, that it will be least effective in textures like the cartilages, which have a sluggish lymph flow. This is probably the reason, or the chief reason, why the cartilages figure more prominently than the fibrous structures in the morbid anatomy of old gouty joints. Probably both tissues were originally infiltrated with biurate crystals in equal degrees; but the fibrous structures afford greater facilities for their re-solution in the periods of amendment than do the cartilages, hence the greater persistence of the deposits in the latter tissue.

The mode in which uric acid produces its injurious effects.—A problem of great interest in the elucidation of gouty manifestations is the mode or modes in which uric acid produces its injurious effects. The main question is, whether these effects are exclusively due to the *mechanical damage* consequent on its precipitation as sodium biurate in the tissues; or whether, in addition, uric acid circulating in the blood in a state of solution is capable of acting as a *chemical poison*.

With regard to the incidents of regular gout, the mechanical theory seems to offer a natural and complete explanation. The crystals of sodium biurate precipitated in the cartilaginous and fibrous structures of the joints necessarily act as foreign bodies; they excite irritation, clog the lymph-channels, exercise pressure on the tissue elements, and impede their nutritive operations. These effects sufficiently account for the inflammation, pain, and swelling which ensue, and explain the remoter degenera-

tive changes which follow after. Nor need we look beyond physical conditions to account for the diversity of the local manifestations. It is easy to understand that depositions occurring within the tense, unyielding structures of the joints would produce results widely different from similar depositions in the loose subcutaneous tissue, or in the rim of the ear. It is equally easy to understand that the suddenness or slowness of the precipitation, its copiousness or scantiness, would necessarily cause great variation in the intensity and character of the local disturbances. So easy and natural is this explanation, that from our general knowledge of pathological cause and effect we might even predict that if similar sudden and copious depositions of crystals of carbonate of lime, or of any other inert substance, were to take place in the same localities, there would follow very much the same train of morbid sequences as are witnessed in connection with uratic precipitations.

It is to the explanation of the phenomena of irregular gout that the mechanical theory of the action of uric acid seems inadequate; and it is to meet this lack that the theory of a poisonous action has been set up and is invoked. The visceral disturbances and manifold neuroses which trouble the gouty have not yet been anatomically traced to uratic precipitation, and there seemed no other way of explaining their occurrence —if they were to be linked with uric acid at all—except by assuming uric acid—or rather the urates—to be possessed of toxic properties. The acceptance of this view appears on several grounds to be extremely difficult.

There is, first, complete absence of direct experimental proof that uric acid is poisonous. Animals have been made to ingest large quantities of uric acid with their food, and urates in solution have been freely injected into their veins, without eliciting any signs of poisoning.

In the next place, the idea that uric acid is poisonous seems opposed to broad biological analogies. Uric acid is the physiological homologue of urea; each of these bodies constitutes, in its separate domain, the final term of nitrogenous metabolism. It cannot be said, without an abuse of terms, that urea is a poisonous substance;[1] and it would be strange if its homologue, uric acid, differed from it in so important a particular as the possession of toxic properties.

The theory appears not less improbable when examined from a nearer point of view. The system of the gouty man is at times surcharged with urates. On the eve of an outbreak the fluids of his body, in parts at least, must be impregnated with biurates to saturation; for, of course, no precipitation can occur until this point is reached. Yet, with fluids thus saturated with urates, such persons often betray not the slightest

[1] The word poisonous is here used with its ordinary meaning. Almost any substance which could be got into solution in the blood in very large quantities would produce deleterious effects, even common salt. Urea can be taken into the stomach in drachm doses without harm; and Dr. Rose Bradford, who has paid particular attention to this point, informs me that the human body in the normal state habitually contains some 30 to 45 grains of urea, and may contain very much larger quantities than this, without provoking any signs of poisoning.

sign of poisoning, and enjoy complete immunity from symptoms of every kind until overtaken, unwarned, by the sudden precipitation which provokes the arthritic attack.

Again, the manifestations of irregular gout are so extremely diverse in seat and character that it is hard to believe that they can be produced by one and the same toxic agent. Sometimes they implicate the stomach, sometimes the liver, heart, or lungs, and, oftenest of all, the nervous system. This diversity is, however, easily explicable on the supposition that the disturbances are caused, not by uric acid in a state of solution acting as a poison, but, like the arthritic manifestations, by uratic deposition; that is to say, by actual precipitation of crystals of biurate into the connective and fibrous structures of the implicated organs, or into the fibrous sheaths of the nerves which control their functions. Observations at the bedside and in the dead-room lead to the inference that uratic precipitation is very variable in its mode and incidence. In certain conditions the crystals appear to descend in sudden and copious showers, which, as in the regular arthritic seizures, provoke a sharp inflammatory reaction. Under other conditions the crystals seem to fall in gentle sprinklings, sufficient perhaps to cause irritation if the implicated tissue be a sensitive one, but not enough to cause downright inflammation. The peculiar pricking pains in the joints which some gouty persons invariably experience after partaking of certain wines are highly suggestive of the occurrence of these slighter precipitations. Now if these slighter precipitations, instead of falling on the joints, were to fall upon the membranes of the brain or upon the fibrous sheaths of the nerve-roots, they would, I submit, afford an adequate explanation of the phenomena of irregular gout. Of course it may be objected that no such slight precipitations have actually been found. But have they been looked for? Has the microscope been used in the investigation? In prosecuting such a search it would have to be borne in mind that precipitations of the kind supposed would be apt to be fugitive, and that negative results would have to be interpreted with caution. For it is obvious, in the case supposed, that when the stress of saturation of the fluids with urates was relaxed, and the blood again recovered its power of dissolving these compounds, these slight deposits would be speedily removed by re-solution, and not a trace of them might remain at the autopsy.

We scarcely realise how imminent a slight but widespread precipitation of the crystalline biurate must not infrequently be in the gouty system—involving the blood and lymph, as well as the fibrous tissues throughout the body. It has been already shown that when the serum of the blood is impregnated with sodium biurate to the extent of 1 part in 6000, supersaturation is attained; and precipitation is then, of course, imminent: now Sir A. Garrod has proved by quantitative analysis that the blood-serum of the gouty man is sometimes actually impregnated with uric acid to this extent. These two facts taken together indicate that the explanation of the phenomena of irregular gout here suggested

stands on a strong basis of *a priori* probability—and thus dispenses with the necessity of assuming that uric acid and its compounds are endowed with poisonous qualities.

This mode of viewing the subject enables us to bring the diverse morbid effects of uric acid into uniform line. Uric acid and its compounds are deleterious simply because of their sparing solubility in the bodily media. It may be said that the final cause of uric acid gravel is the sparing solubility of free uric acid in urine; in like manner it may be said that the final cause of gouty precipitations is the sparing solubility of sodium biurate in blood-serum, lymph, and synovia.

Ætiology.—The conditions which engender, or tend to engender, the gouty state may be divided into those which are inherent in the individual, or intrinsic causes, and those which operate from without, or extrinsic causes.

The intrinsic factors include age, sex, heredity, bodily conformation, and individual peculiarities.

Age.—Gout is in the main a disease of middle life and advancing years. Typical arthritic attacks usually first show themselves between thirty-five and fifty. Where there is a strong hereditary tendency the onset is often earlier—and young men and even boys at school may undergo typical joint attacks. On the other hand, the first manifestation of gout may be postponed to old age; in these cases the complaint is usually of a mild character, and appears almost as if it were an incident of senescence. It not infrequently happens that gouty attacks show themselves in greatest frequency and severity in the fourth and fifth decades of life, and that the subsequent decades are comparatively or entirely free from such attacks.

Sex.—Gout is much more common among men than women. Indeed typical gouty paroxysms are rare in the female sex. The difference, no doubt, is due in great part to the different dietetic habits of the two sexes. It is, moreover, held that the catamenial discharges to a certain extent protect women against gout. After the menopause symptoms of irregular gout are not uncommon, especially in women of plethoric habit.

Heredity.—Gout runs strongly in families. Fully three-fourths of cases of gout occurring among the easy classes can be traced back distinctly to a gouty ancestry. It is noteworthy that although the women of gouty families may escape overt gout, the transmission of the disease to offspring is more certain through the female than through the male line. A grandson may inherit gout in full force from a gouty grandfather through a mother who has never herself exhibited any gouty manifestation. It is also beyond doubt that gout newly acquired during life by the action of extrinsic causes may be transmitted to the children. It is significant that in such cases the gouty taint shows itself in the later born and not in the earlier born children. The history of gout supplies some important instances of the possibility, so much debated among biologists, of the hereditary transmission of acquired characters.

Bodily conformation and individual peculiarities.—Persons of large frame and vigorous appetite with a tendency to corpulence may be said to have a natural proclivity to gout; and they easily become overtly gouty if they fall under the operation of the dietetic factors and the sedentary habits which favour the invasion of the complaint.

The extrinsic causes of gout include errors of eating and drinking, idle and sedentary habits of life, lead impregnation, and renal disease.

Food and Drink.—Dietetic habits play a large part in the production of gout. Gout is the nemesis of high living. Of solid food the red meats and game are held to be more promotive of gout than the white meats and fish. Highly nitrogenised articles of food favour an excessive formation of uric acid, and therefore favour the accumulation of urates in the blood. Speaking broadly, articles of food of animal origin are richer in nitrogen than those of vegetable origin. They are also more attractive to the palate, and for that reason tempt to gluttonous feeding. The upper classes—who in this country eat meat two or three times a day—provide the largest contingent to the company of the gouty. On the other hand, agricultural labourers and unskilled workmen, who rarely get meat more than once a day, and often only three or four times a week, enjoy almost complete immunity from the ordinary forms of gout.

Of all articles of diet, however, the most provocative of gout are alcoholic beverages. Sir Alfred Garrod expresses himself very strongly on this point. He writes: "There is no truth in medicine better established than the fact that the use of fermented liquors is the most powerful of all the predisposing causes of gout; nay, so powerful, that it may be a question whether gout would ever have been known to mankind had such beverages not been indulged in."

Alcoholic liquors are, however, not all equally potent in fostering the gouty habit; and the difference between them in this respect has comparatively little reference to their percentage of alcohol. Port, sherry, Madeira, Burgundy, strong ales, porter, and stout are much more powerful factors in the production of gout than distilled spirits. The prevalence of gout in different districts and countries is found to have a close relation to the kind of alcoholic beverage in common use. In Scotland and Ireland, where whisky is the common drink, gout is not so prevalent as in England, where malt liquors are the popular beverages. In France gout is most common in the Burgundy district; while in the Rhenish provinces of Germany—where various classes of hock are consumed—gout is but little known. Champagnes, especially the sweeter sorts, are undoubtedly gouty wines; cider, on the other hand, has not much power in this direction. It has to be remembered that those who habitually use the richer wines and the heavier kinds of malt liquors are also often large consumers of meat, and that these two factors reinforce one another in the production of gout. Hence certain occupations conduce to gout; and butlers, gentlemen's servants, butchers, innkeepers, brewers' draymen and

cellarmen are frequent victims to this complaint. It may further be observed that—apart from quality and quantity—the mode of using alcoholic beverages has much to do with their efficiency as producers of gout. Gout is but rarely found among drunkards, topers, and loafing beer-swillers; nor is it a special concomitant of cirrhosis of the liver, nor of other organic tissue changes which originate from alcoholism. Gout is rather an incident of the legitimate dietetic use of alcoholic beverages. Persons who acquire gout as a consequence of their dietetic habits are, as a rule, above reproach in regard to their sobriety; but, at the same time, it cannot be gainsaid that their scale of living is habitually high, and that they are of the number of those who "fare sumptuously every day."

The dietetic habits which dispose to gout have their incidence mitigated on the one hand, or intensified on the other, by the general mode of life. Those who lead an active out-of-door existence can carry off with impunity a scale of feeding which involves those of more sedentary and studious habits in the pains and penalties of gout.

Lead impregnation and gout.—It is well known that painters and plumbers are liable to gouty manifestations. The association of gout with plumbism is very marked in the metropolitan area. Among hospital patients in London, Sir A. Garrod found that 33 per cent of those affected with gout showed distinct evidence of having been poisoned with lead. Sir Dyce Duckworth observed that of 136 cases of unequivocal gout among out-patients of both sexes, 25 presented signs of lead impregnation. All these were males, and followed the occupation of plumbers, painters, compositors, or workers in lead-mills. This association is much less frequently observed in Scotland and the North of England than in London. Dr. Oliver of Newcastle (where a large number of persons are employed in lead-works) remarks: "We do not see in the north that intimate relationship between gout and saturnine poisoning. Workmen from the south develop it in the North of England. The natives of the north, though equally exposed, seldom become gouty even when the kidneys are affected" (5). [*Vide* art. "Lead Poisoning," vol. ii. p. 962.] Sir Dyce Duckworth cites an analysis made by Frerichs of 163 cases of lead poisoning in the hospitals of Berlin. Among these there was not a single case of true gout. The aggregate of the evidence collected on this point goes to show that the association of lead poisoning with gout is scarcely noticeable except in a population among whom gout from other causes is prevalent, and that lead impregnation can rarely promote gout except in persons who have already either a hereditary or an acquired predisposition to the complaint.

Immediate causes of a fit of gout.—The immediate occasion of a gouty outbreak is often undiscoverable. The outbreak seems to arise in most instances as the culminating effect of the predisposing factors, and often occurs unexpectedly in the midst of apparent health. The spring and autumn seasons appear favourable to an outbreak. Not infrequently, however, some particular incident or circumstance can be distinctly

indicated as the determining cause of the attack. It may be an unusual access of luxurious living or of indulgence in rich wines; sometimes the attack is traceable to some special worry or anxiety, or a fit of anger, or studious effort. Exposure to cold is sometimes the immediate antecedent of a paroxysm; sometimes an accidental injury to a member or a joint lights up the mischief. Whatever the immediate determining cause of a fit of the gout may be, or appear to be, it is obvious that the incriminated causal factor could not have taken effect had not the sufferer been already gouty and constitutionally disposed and ripe for an attack. The occurrence of a fit of the gout is, therefore, not infrequently a demonstration of the pre-existence of a long-suspected condition of latent gout.

Connection with other diseases.—As gouty persons advance in life they sometimes become glycosuric. This circumstance does not usually appear to aggravate their condition materially. Gouty glycosuria rarely proves severe; it is, as a rule, unaccompanied by thirst and diuresis, and is efficiently controlled by moderate dietetic restrictions. In splenic leukæmia the quantity of uric acid produced and voided with the urine is greatly increased; and it might have been expected that persons so affected would exhibit a strong proclivity to gout. However this does not appear to be the case. I have only encountered one example in which the two diseases were associated; and in that case the patient had been a martyr to gout for very many years before the spleen became enlarged and the blood leukæmic. Sir Dyce Duckworth observed two instances in which gout coincided with splenic leukæmia; in one case the gout was obviously long antecedent to the splenic enlargement; in the other the sequence was doubtful. I am not aware that any case has been recorded in which gout supervened as a clear consequence and result of splenic leukæmia. Certain it is that patients with splenic leukæmia may go on voiding enormous quantities of uric acid for years without showing the least indication of gout.

Uric acid gravel and calculus are sometimes associated with the gouty diathesis: the connection between these two conditions is however by no means close. The great majority of gouty persons never suffer from urinary gravel; and, conversely, only a small percentage of the subjects of uric acid gravel suffer from gout. The geographical distribution of the two complaints is widely different. In Scotland, where gout is rare, stone is comparatively common. Mr. Plowright has shown that there is no correspondence between the prevalence of gout and the prevalence of stone in the several counties of England. Some of the counties which have a high mortality from gout have a low mortality from stone. The county of Norfolk, which is the chief stone district of England, is comparatively free from gout. Uric acid gravel and calculus are common in natives of India, among whom gout is practically unknown.

The relation of gout to disease of the kidneys has already been discussed (see Morbid Anatomy, p. 166).

Diagnosis.—Articular gout is liable to be confounded with rheumatism —acute and chronic; with rheumatoid arthritis; and, more rarely, with gonorrhœal, pyæmic, or traumatic synovitis.

As a rule the diagnosis of acute articular gout is easy; but exceptional cases of difficulty occur. The gouty character of the inflammation is affirmed by the discovery of uratic concretions in the rim of the ear or elsewhere. Gouty paroxysms are usually recurrent; and the attack is either inexplicable, or is traceable to dietetic errors or mental worry, but very rarely to cold. Rheumatic attacks, on the other hand, are nearly always traceable to some distinct exposure to cold. The family history of the patient often yields important information. Gout runs strongly in families; and if inquiry discloses that the ancestors were subject to gout, that fact, in doubtful cases, is highly significant. Of almost equal significance are the past mode of life and the dietetic habits of the patient, especially as regards the use of alcoholic beverages.

Age and sex have an important bearing on the diagnosis. Acute gout is not often seen before the age of thirty or thirty-five, unless the hereditary tendency be very pronounced. Acute rheumatism, on the contrary, is most common in early life between the ages of fourteen and thirty. The female sex is very markedly more exempt from gout than from rheumatism.

The clinical characters of the symptoms in gout and rheumatism are usually quite distinctive. In gout the febrile movement is less intense, while the local pain is more severe; the larger joints (except the knees) are less generally implicated, and there is an absence of cardiac complications. Severe sweating is not nearly so conspicuous in gout as in acute rheumatism. When the attack is subsiding œdematous pitting is usually observed in gout about the swollen joints, and is followed by desquamation of the cuticle. These signs are not observed after rheumatic inflammation.

It is in cases of chronic and irregular gout that the diagnostic difficulties are most embarrassing. The convenient term *rheumatic gout* is no longer in favour; and, no doubt, cases so designated are in the majority of instances examples either of pure gout or of pure rheumatoid arthritis. Nevertheless it may be said that in exceptional cases the two conditions are combined, and to such cases the name rheumatic gout may be correctly applied. There is no obvious reason why gouty subjects, especially those of the asthenic type, should be exempt from rheumatic and rheumatoid affections; and cases occur, more commonly among women than men, where the combination of symptoms and physical signs indicates an overlapping of the two morbid conditions, and where the designation of rheumatic gout is strictly appropriate.

Prognosis.—The prospect of survivorship in gout depends largely on the presence or absence of complications. Members of gouty families are often long-lived; and if the kidneys remain sound, and the periodical attacks are not too frequent and too protracted, the general level of health is not appreciably lowered. The appearance of albumin in the urine is

always an untoward sign in gout, because it too often portends the implication of the kidneys in the gouty process. It is, however, important to be aware that gouty persons may have traces of albumin in the urine for many years, even to old age, without falling into genuine Bright's disease.

Therapeutics of gout.—The treatment of gout ranges over a wide field—according to the phases of the complaint, the kind and nature of the local manifestations, and the age, temperament, and antecedents of the patient. Attention will be directed first to the treatment of the gouty paroxysm; in the second place the general management of the gouty state will be considered—the means to be adopted to prevent or lessen the frequency and severity of the recurrent attacks, and to relieve the irregular manifestations of the diathesis.

Treatment of the gouty paroxysm.—The treatment of acute gout is conducted, in the main, on the same lines as that of other inflammatory ailments. At the outset the bowels are relieved by a suitable purgative: if the fever run high, the patient is confined to bed; the inflamed joints are kept at rest; the diet should consist of milk and farinaceous articles; and diluents should be freely administered. It is necessary, however, to bear in mind the specific character of the inflammation. Gouty patients bear bleeding badly, and neither venesection nor the application of leeches is to be recommended. The application of cold to the inflamed joints must be strongly deprecated: such a proceeding involves serious risk of metastasis of the morbid process to the internal organs. The joints should be simply swathed in cotton wadding covered over with oil-silk. This appliance acts as a light poultice, and promotes cutaneous transpiration and a kindly development of the tumefaction, which is usually followed by great relief of pain. Repeated purgation is undesirable; it is of more service to encourage the action of the skin and kidneys than to induce watery intestinal discharges. During the inflammatory period the use of meat should be avoided; there seems no doubt that neglect of this precaution tends to prolong the attack, and adequate nourishment of a less stimulating character can be supplied by means of milk, bread, farinaceous puddings, and a little fish. Alcoholic beverages, except under special circumstances, should be withheld during the febrile period. In weak or elderly persons, whose systems have been permanently lowered by repeated attacks, the diet should not be unduly meagre; nourishing soups, white meats, and a modicum of alcoholic stimulants should be allowed. In sthenic cases, during the febrile period, the acidity of the urine should be controlled and its volume increased by the systematic exhibition of bicarbonate or citrate of potash. Salines containing soda should, for chemical reasons, be avoided. If a saline aperient be thought desirable, the sulphate of magnesium should be chosen. The occasional use of mercurials for the relief of hepatic congestion is advisable. When the nights are very restless a Dover's powder, or other opiate, may be administered at bed-time.

For the purpose of controlling the gouty inflammation and shortening

the attack there is no remedy comparable with colchicum. Ten to twenty-five drops of the wine or tincture may be given two or three times a day, or a grain of the extract may be given at bed-time in pill with a few grains of Dover's powder. The use of colchicum requires care; some persons are exceptionally sensitive to the action of the drug, and experience a sense of faintness from heart failure unless the dose be very small. In other persons ordinary doses of colchicum induce purging, and the dose must be reduced accordingly. The striking effect of colchicum in reducing gouty inflammation, and in diminishing the pain, is often very remarkable, and is, undoubtedly, of a specific character. Fears have been expressed that, although the immediate action of colchicum be favourable, the more remote effects are not salutary, and that the use of the drug tends to increase the frequency of the recurrence of the paroxysms. These apprehensions do not, however, appear to rest on any solid ground of facts.

In cases where colchicum proves ineffective, or is badly borne, iodide and bromide of potassium, salicine, the salicylates, and the salts of lithia may be used.

When convalescence is fairly established the stiffened and swollen joints should be cautiously massaged and gently exercised; the patient should be encouraged to take the air; the dietetic restrictions should be gradually and tentatively removed; nux vomica, mineral acids, or other suitable tonics should be administered; and, finally, change of air to the sea-side or to an upland station is to be recommended, as helpful to accelerate the restoration to health.

Treatment of chronic and irregular gout—the general management of the gouty state.—The fundamental aim of a rational treatment of chronic gout must always be to diminish the incidence of the diathesis on the constitution. According to the view set forth in this article the essence of the gouty diathesis consists in an enduring tendency to the accumulation of urates in the bodily fluids and to their precipitation as crystals of sodium biurate in the tissues.

The means we possess of influencing the factors which govern the precipitation of sodium biurate in the body may be divided into those which belong to the domain of diet and regimen, and those which consist in the administration of medicinal substances.

Diet and regimen.—It has been shown that one of the main factors in determining uratic precipitation is the percentage of urates in the medium. Other things being equal, the larger the proportion of urates present, the earlier and more abundant will be the deposition of the crystalline biurate. Our power of controlling this factor lies almost entirely in the direction of regulation of the diet. Numerous series of experiments have been made on the effect of diverse kinds of food on the production and excretion of uric acid. The point of chief therapeutical interest which has been clearly made out is this: that the ingestion of large quantities of proteid matter is attended with an increased production of uric acid, and *vice versa.* It does not appear clear that proteid substances derived from the animal

kingdom differ in this respect from those derived from the vegetable kingdom. Nevertheless, inasmuch as the commonly-used articles of food of animal origin—such as butcher's meat, poultry, game, fish, eggs, and cheese—are richer in proteid stuff than the commonly-used articles of vegetable origin — such as bread, oatmeal, rice, potatoes, and garden produce—it is true that a vegetable diet is less productive of uric acid than an animal diet. The most trustworthy experiments indicate that fat, starch, and sugar have not the least direct influence on the production of uric acid; but as the free consumption of these articles naturally operates to restrict the intake of nitrogenous food, their use has indirectly the effect of diminishing the average production of uric acid. There may be, and indeed undoubtedly are, other differences between animal and vegetable articles of food, and between one article and another of the same class, which are highly important. Articles of diet differ considerably among themselves in their digestibility and in their stimulating qualities; but in regard to the point under notice, namely, their direct influence on the production of uric acid, articles of diet must, so far as our present knowledge goes, be classified according to the percentage of albuminoid matters contained in them. As a rough guide in the choice of food for the gouty, the subjoined table may prove useful:—

TABLE showing the Average Percentage of Albuminoid Matters contained in diverse Articles of Food.

Animal Food.	Albuminoid Matter.	Vegetable Food.	Albuminoid Matter.
Butcher's meat .	19 per cent	Bread . . .	8 per cent
Fowl . . .	20 ,,	Oatmeal . .	12 ,,
Game . . .	22 ,,	Rice . . .	6 ,,
Fish . . .	17 ,,	Green peas . .	6 ,,
Egg . . .	13 ,,	Potatoes . .	2 ,,
Milk . . .	4 ,,	Carrots and turnips	1 to 2 ,,
Cheese . .	30 ,,	Green vegetables and salads .	1 to 2 ,,
		Fresh fruit (excluding nuts) .	0·5 to 1 ,,

In choosing a diet for persons disposed to uratic precipitations, regard must be had of course to the whole condition, and especially to the peculiarities of the individual. Nowhere, perhaps, is it more necessary than in gout to consider the man as well as the ailment, and very often more the man than the ailment; nevertheless the general rule of diet is, I think, pretty clear. Gouty people should be advised to partake cautiously of butcher's meat, fowl, game, and cheese—and to partake as freely as their digestion will permit of bread, rice, garden vegetables, salads, and fruit. The advantage to be gained from an adjustment of the dietary on these lines may be inconsiderable, or even inappreciable, in

cases of inveterate gout; but it may be of critical moment in the slighter cases. A diminution of one or two grains per day in the amount of urates thrown into the circulation may make all the difference between the occurrence or non-occurrence of an arthritic attack.

Alcoholic beverages.—The highly-important part played by certain kinds of alcoholic beverages in the genesis of the gouty constitution, and in fostering a proclivity to uratic precipitation, has been well established. The precise mode in which this adverse influence takes effect is not well understood. The most trustworthy researches indicate that these beverages, in their legitimate use, exercise no appreciable direct influence on the quantity of uric acid produced in the body. Nor is their action proportionate to the percentage of alcohol contained in them. Distilled spirits have but little influence in promoting gout, and whisky and gin less than brandy. On the other hand, the richer wines—port, sherry, Madeira, champagne, Burgundy—and strong ales and stout are highly provocative of gouty manifestations. The most wholesome wines for the gouty are clarets and hocks. Fully fermented, mature, and dry wines are less injurious than sweet and new wines. In gouty persons with a robust habit of body a regimen of total abstinence from alcoholic stimulants is generally the best. Practical experience, however, shows that hard and fast lines on this matter cannot be maintained. A good many gouty persons, as a matter of fact, fare better with a moderate allowance of alcoholic liquors than without any. With some cases of this class the complete withholding of stimulants fosters a low asthenic type of gouty manifestations, which are more injurious to the general health than frank inflammatory attacks. The personal experience of an observant patient in regard to the point under consideration should always be elicited and carefully weighed by the medical adviser. Such experience not infrequently furnishes valuable indications which may be turned to good account in the general management of the case.

The use of common salt.—It has already been pointed out that all sodium salts act very adversely on the solubility of sodium biurate, and greatly promote its precipitation. It has also been shown that the topographical distribution of uratic deposits through the body bears a striking relation to the percentage of sodium salts contained in the several organs and tissues. In fact, the chemical evidence before adduced seems to point to the deduction, that if we possessed the power of regulating the proportion of sodium salts in the fluids and tissues of the system, we should be able effectively to control the occurrence of uratic precipitations. Our power in this respect, however, is restricted: sodium salts belong to the physiological constants of the blood, and their proportion therein can only be altered through a limited range. These remarks apply especially to the most abundant of them, the sodium chloride. It has been found, in experiments on animals, that when common salt is given in excess with the food, or injected into the veins, the surplus is for the most part quickly removed by the kidneys, and thus increase of its percentage in the blood is but small and transient. And, conversely,

when animals are fed with food abnormally poor in salt, there is but a slight falling-off in its proportion in the blood. The blood clings with great tenacity to its proper percentage of sodium chloride; and the experimental evidence indicates that in case of a threatened salt-famine within the economy the blood has the faculty of supplying its necessities by extracting salt from the less vital fluids and tissues: contrariwise, in case of a glut of salt in the blood, the overplus is temporarily passed over into the serous cavities until such time as the kidneys have succeeded in restoring the normal equilibrium. These observations lead to the inference that by lessening the intake of salt with the food we should only abate its proportion in the blood to a slight degree, but should diminish its proportion in the synovial fluids and fibrous tissues considerably. Acting on these opinions, I have been in the habit for some years past of advising gouty patients to restrict their use of sodium chloride as a condiment, and to substitute for it, as far as practicable, the use of potassium chloride. Speaking from a considerable experience, I can affirm that the potassium chloride acts beneficially on the gouty system; it is as harmless as common salt, and not unlike it in flavour; it may be used dietetically the year round as a table salt, either alone or mixed with a fourth part of common salt, or it may be administered in solution in water in the dose of ten to twenty grains three times a day. I have seen such a plan of treatment followed out for years, apparently with the effect of preventing or diminishing the recurrence of arthritic attacks, and of removing the various manifestations of irregular gout.

Administration of medicinal substances.—The medicinal agents which have been chiefly employed in the treatment of gout, with a view of controlling the tendency to uratic precipitation, are the alkaline carbonates, the salicylates, the carbonate of lithia, piperazine, and the waters of mineral springs.

Alkalies.—Alkaline substances are largely employed in the treatment of gout, both as pharmaceutical preparations and as components of mineral waters. It is widely believed that the alkaline carbonates administered internally, by increasing the alkalescence of the blood, enhance its solvent power on the material of gouty deposits, and thereby delay or prevent their formation. The experimental evidence before adduced entirely destroys this hypothesis. It has been conclusively proved that alkalescence, as such, has no influence whatever on the solubility of sodium biurate. It has, moreover, been shown that the addition of an alkaline carbonate to blood-serum impregnated with uric acid produces no appreciable effect on the process of maturation, or on the advent of precipitation of the crystalline biurate in the medium. The use of alkalies in gout has been advocated on another ground. It is held, in a vague sort of way, that there is an undue prevalence of acid in the gouty system, and that the blood is less alkaline than it should be. In some quarters it is even believed that this is the primary vice of the gouty state, and that there exists a so-called "acid dyscrasia" which dominates the whole

condition. I have been at some pains to ascertain what foundation there is for this belief, and have found very little of any kind, and none that is really valid. In the numerous examinations of the blood in gouty subjects made by Sir Alfred Garrod, the serum was invariably found to be alkaline, never acid or even neutral. But he remarks that there is often (not always) a marked alteration in the degree of its alkalinity, and that in cases of chronic gout the serum sometimes shows a near approach to neutrality. It is, however, obvious that observations on the alkalinity of the blood have no validity in regard to the point under consideration unless they are made on cases of gout pure and simple. Gout is often complicated not only with pyrexia, but with serious secondary lesions in the kidneys and joints, which lead to a profound cachexia. These secondary lesions bring with them blood changes of their own, which are but remotely connected with the primary disorder, and have no bearing on the ætiology of uratic precipitation. In the last few years some exact quantitative measurements have been made of the alkalinity of the blood both in health and in disease (6, 7). These researches indicate that a diminished alkalescence of the blood is a common pathological deviation, and that it occurs in a variety of conditions which have no relation to gout —namely, in pyrexia, diabetes, carcinoma, acute rheumatism, anæmia, leukæmia, and apparently in every kind of general cachexia. These facts and considerations suffice to show that, in the present state of our knowledge, the belief in an acid dyscrasia in gout rests on a pure assumption.

Lastly, the use of alkalies in gout is advocated on the ground that they facilitate the task of the kidneys in separating uric acid from the blood. The experimental evidence hitherto adduced on this point is so hopelessly contradictory that no conclusion can be safely deduced from it.

Clinical experience on the use of alkalies in gout speaks with a doubtful voice. I think I may say that few practitioners have employed alkaline remedies in gout with more determination than myself. In years gone by I have repeatedly administered the bicarbonate and citrate of potash continuously for three and four years, and in sufficient doses to maintain the urine persistently alkaline, yet in these very cases I have seen the arthritic attacks recur with apparently unabated regularity.

Salicylates.—In recent years the salicylates have been largely tried in the treatment of gout, both in this country and on the Continent. The clinical evidence as to their efficacy is very contradictory. The use of the salicylate of soda is open to the same objections as lie against the use of all soda salts. The same objections do not apply to the salicylates of lithia and potash.

Salts of lithia and piperazine. — Lithium salts were first introduced into medical practice by Sir Alfred Garrod, and they are now extensively employed in the treatment of chronic gout. The preparations most commonly employed are the carbonate and citrate, which are given either simply dissolved in water or in the guise of an effervescent water, as "lithia water," containing five to ten grains in the pint. Piperazine is a more recent introduction, and is administered in the same ways and in

similar doses. Carbonate of lithia and piperazine have been recommended for the treatment of gout expressly on chemical grounds. Watery solutions of these substances possess a considerable solvent power on free uric acid; and it has been inferred from this fact that their administration internally might exercise a favouring influence on the solubility of sodium biurate in the bodily fluids, and thereby tend to prevent the formation of uratic deposits. This inference does not, however, appear to be justified, because the solvent relations of free uric acid and of sodium biurate are widely different. It was found experimentally that the addition of carbonate of lithia or piperazine, in the proportion of 0·1 per cent and 0·2 per cent, to blood-serum or synovia, had not the slightest effect in enhancing the solvent power of these media on sodium biurate, nor the slightest effect in retarding its precipitation from serum and synovia artificially impregnated with uric acid. If these bodies have any beneficial action in gout it is certainly not due, as has been supposed, to their solvent action on the material of gouty concretions.

Mineral Springs.—The facts set forth in the preceding portion of this article respecting the solvent relations of sodium biurate have an important bearing on the use of mineral springs in the treatment of gout. A considerable number of the springs to which gouty patients resort are strongly impregnated with sodium salts; and it has been conclusively shown that all the salts of sodium exercise an adverse influence on the solubility of sodium biurate, and hasten its precipitation. It is not, therefore, surprising to learn that not infrequently the first effect of these waters on a gouty patient is either to provoke a downright attack of gout, or to aggravate the symptoms under which he was suffering. This event is now recognised by many physicians practising at these spas as a thing to be looked for; and experience has taught them the necessity of caution in regard to the quantity of the waters to be taken by new-comers. They comfort themselves and their patients, however, with the assurance that this preliminary storm is a necessary prelude to the calm amendment which is to follow. There is, no doubt, some foundation for this idea. It is no fiction that a gouty man, tormented by symptoms of irregular gout, is relieved by a regular arthritic attack. It may be presumed that this relief arises from the complete, or approximately complete, precipitation into the structures of the joints of the urates floating in his blood and lymph. The urates are thereby almost as effectually removed from the vital fluids as if they were eliminated by the kidneys. But it must, I think, be allowed that this is a rough mode of cure, and that it brings with it serious pains and perils of its own. My impression is that gouty persons should either entirely avoid springs which owe their activity to sodium salts, or should use them very sparingly; it is difficult to believe that they can do any direct good, and easy to believe that they can do direct harm. If they do any good at all it must be indirectly, by acting on the liver and the intestinal tract.

There are, however, other springs of high and growing repute in the

treatment of gout which are not open to these objections. These springs contain no soda, or traces only; and the sum of their mineral constituents does not exceed that which is often present in ordinary potable waters. They contain for their principal ingredient a little carbonate or sulphate of lime; and it is very doubtful whether the whole of this is absorbed into the blood: most of it probably passes out inertly with the fæces. In fact, springs of this class may practically be considered as equivalent to ordinary drinking-water, except that several of them have the advantage of being thermal. Among springs of this class may be mentioned: in our own country, the waters of Buxton, Bath, and Strathpeffer; in Germany, the waters of Gastein, Wildbad, Pfeffers, and the Sauerling spring at Carlsbad; in France, the waters of Aix-les-Bains, Contrexéville, Vittel, and Barèges. Now there can be no reasonable doubt that the efficacy of these springs has nothing to do with their scanty mineral ingredients, but depends essentially on their watery constituent. They are drunk freely, and on an empty stomach. Their action would be to dilute the blood temporarily, and lower its percentage of urates and sodium salts. This effect would tend to retard or prevent uratic precipitation, and thus give the defective kidneys additional time to overtake their arrears in the task of eliminating uric acid.

It may be asked whether the drinking of water at home would not answer as well as resorting to a mineral spring. The inference from the foregoing observations is that, other things being equal, the beneficial results would be the same. But the "other things" never are "equal." It would scarcely be practicable for a man going about his usual business to drink eight or ten tumblers of water on an empty stomach every day for two or three weeks: at a watering-place the visitor has nothing else to do than to attend to his "cure." Moreover, in getting away from home the invalid leaves behind him the worries of his daily life, and has the advantage of change of air and scene, a salutary modification of diet, and abundant leisure for outdoor exercise. These collateral influences help to raise the general level of health, and quicken the action of the secretory cells. Besides, the waters of mineral springs are not merely used in the way of drinking; they are also used in the way of baths and various kinds of douches; and the proper hydrotherapeutic treatment is usually supplemented by shampooing, passive motion of the stiffened joints, electric baths, hot packs, and other appliances, all of which are of great service in chronic gout. I do not think, therefore, that gouty patients, if they can afford the time and expense, should forego the advantages of the time-honoured practice of a visit to a mineral spring. At the same time, a word may be said in favour of a more systematic use of water in the everyday life of the gouty. I have observed that some gouty persons are very sparing in their use of diluents; such persons should be encouraged to be habitually more liberal in this respect. In a few cases it might even be possible to imitate, with plain water, the regular two or three weeks' course at the spa, and to repeat this course twice or thrice a year, as a prophylactic measure.

General hygienic management.—Sedentary occupations and idle habits of life are highly injurious to gouty persons, and tend strongly to provoke recurrent arthritic attacks, and to engender the various irregular manifestations of the disease. Gouty patients should be encouraged to lead an active out-door life; they should take exercise systematically in the open air in the way of walking, horse-riding, or driving; and should, as far as practicable, avoid heavy dinners and late hours. Due foresight should be exercised to evade anxious and worrying engagements, and to guard against undue mental or bodily fatigue. The body should be clothed in light flannels, and the action of the skin promoted by the use of baths and friction. Gouty men are often disposed to be neurotic, and torment themselves with vain ponderings over their symptoms. It is not, therefore, desirable, unless there be some special reasons, for them to relinquish their occupations and lapse into a life of idleness. Some persons adopt the practice of taking a tumbler of hot water, or some hot diluent, on going to bed at night and on rising in the morning. I believe that this practice is a wholesome one for gouty constitutions, and tends to clear the blood of uratic impurities.

Local treatm nt of gouty affections.—As a rule the local manifestations of chronic gout are most effectively combated by means directed to diminish the general incidence of the gouty state, but sometimes they call for separate treatment. The thickening, stiffening, and aching about gouty joints often persist long after the inflammatory movement has passed away. In this condition relief may be given by topical measures. Among the means most in vogue for this purpose are:—Painting the joints with iodine, persevering use of wet compresses, friction with stimulating liniments, shampooing and passive motion, and the application of flying blisters. Hot mineral baths, douches, and galvanic baths have also been employed in such cases with conspicuous success. Subcutaneous tophi in the neighbourhood of joints sometimes become tense and painful, and interfere with the movements of the adjacent articulations. It has been asserted that the persevering application of pledgets soaked in solutions of carbonate of lithia or carbonate of potash to such enlargements has some effect in reducing their size and in dissolving out the urates contained in them. It is not desirable to puncture or cut these swellings, as such procedures are very apt to leave behind them indolent sores which are very difficult to heal. Sometimes such tophi burst spontaneously, and form indolent ulcers, which occasionally penetrate into the joints, and discharge a purulent sanies mixed with uratic crystals. The most effective way of dealing with these ulcers is to immerse the part for some hours daily in a large can of warm water, with a view of dissolving out the offending urates. I have seen a similar mode of treatment afford great relief in cases where uratic deposits encrusted the heels or palms, thereby causing pain and hindering locomotion.

It is not always desirable to interfere too actively with gouty affections of the skin; they often appear to operate as a sort of safety-valve to the

gouty system; but sometimes the itching and irritation caused by them become well-nigh intolerable, and seriously interfere with sleep and the general well-being of the patient. In such cases constitutional measures must be eked out by local means. Solution of borax with glycerine, boracic acid in vaseline, lead lotion, zinc ointment, bismuth and starch powder applied to patches of gouty psoriasis, eczema, or prurigo often afford great relief. In the case of dry eruptions, associated with much itching, I have found the solid paraffin a valuable resource. A piece of hard paraffin of the size of the thumb is pared down to a smooth surface with a penknife, and the itching surface is rubbed over morning and evening with this, so as to leave a delicate coating of paraffin on the skin. It is a perfectly cleanly and wholly invisible application; and probably acts by protecting the cutaneous surface against the contact of the atmosphere.

WILLIAM ROBERTS.

REFERENCES

1. FAGGE and PYE-SMITH's *Handbook of Medicine*, p. 674. — 2. FRERICHS. R. Wagner's *Handwörterbuch d. Physiol.* Bd. iii. Part 1, p. 463. — 3. GARROD, A. Reynolds' *System of Medicine*, vol. i. p. 817.—4. LEVISON, Dr. F. *Zeitsch. f. klin. Med.* Bd. xxvi. Heft. 3 u. 4.—5. OLIVER, Dr. *Goulstonian Lectures on Lead Poisoning*, 1891, p. 102. — 6. PEIPER. *Virchow's Archiv*, 1889. — 7. RUMPF. *Centralblatt f. klin. Med.* 1891.

W. R.

DIABETES MELLITUS

SYNONYMS.—*Diabetes; Saccharine diabetes; Glycosuria; Mellituria; Diarrhœa urinosa; Polyuria.*

Definition. — A constitutional disease characterised by the persistent passage of glucose in the urine.

History.—In the papyrus Ebers, which is a copy of an Egyptian medical compilation already old in the time of Moses, there is mention of polyuria, and it is hard to conceive that such a marked departure from health could at any time have escaped observation; yet no notice of it is to be found in Greek writings earlier than those of Aretæus of Cappadocia (*circa* A.D. 150), who is supposed to have been a contemporary of Galen, and was probably also a Roman physician. His description, like that of the unknown Egyptian priest, or that of Galen, who wrote at length upon the disease, lays stress only on the polyuria and thirst. The important fact that the urine contains sugar appears to have escaped the notice of all early European writers; nevertheless, if we may trust some passages of the Ayur Veda (*circa* A.D. 500) it was known at that time to the Hindus. In a Cingalese writing of the fifteenth

century diabetes is undoubtedly referred to as "madu mehe," or *honey urine*, so that in this respect European knowledge lagged sadly behind; for it was not until 1679 that our countryman Willis mentioned that the urine of diabetes has a sweet taste. A century later Dobson of Liverpool demonstrated the presence of sugar, which discovery so impressed Cullen, that he would hardly allow that this was not true of all cases of diabetes; and not until the present century has the distinction been firmly established which we recognise in the names *diabetes insipidus* and *diabetes mellitus*.

Etiology.—The mean annual mortality from diabetes mellitus in Europe does not exceed 5 per 100,000 of persons living. But within this area considerable variations may be noted: for example, in Paris[1] the figure is as high as 14; in Malta 13; in Copenhagen 7; in London 7; in Vienna 4, and in Naples 3. In the whole of England it is 7, in Ireland 3, in Scotland 2, in Norway 2, in Prussia 2, and in Italy 1·5. Diabetes is said to be extremely common among the educated and commercial classes of natives in India and Ceylon, while it is unknown among the Chinese and Japanese, and the negroes of Africa. This immunity of the negro appears to persist, partially, at least, in America; for Prof. Tyson of Philadelphia has stated that he has never met with a case in the negro, although he has modified the inference to be drawn from his personal experience by informing me that he has since heard of two examples in the practice of a coloured physician.

American authors claim that their national statistics prove the annual rate of mortality from diabetes for all races to be much lower in the States than in Europe; it will be interesting to see whether this

[1] DEATHS FROM DIABETES IN PARIS (Bertillon).

Year.	Number of Deaths from Diabetes.	Proportion per 100,000 Population.
1888	291	13
1889	307	14
1890	298	13
1891	311	13
1892	295	12
1893	349	14
1894 (8 months)	209	..

ENGLAND—WALES.

Year.	Deaths from Diabetes.	Total Deaths.	Proportion per 10,000 Deaths.	Proportion per 100,000 Living.
1882	1253	516,654	24·4	4·7
1892	2011	559,684	35·9	7·0
LONDON.				
1892	292	86,833	33·9	7·0

superiority is maintained as the condition of the large towns becomes more and more comparable with that existing in Europe; and especially when their municipal statistics shall have been placed upon an equally satisfactory basis.

In France the disease is said to prevail extensively in Normandy; in Italy it is apparently more common in Tuscany than in other parts of the kingdom.

Scudamore was certainly mistaken in the assertion that the Scottish people are specially liable to diabetes; and his unsupported statement, completely contradicted as it is by the Registrar-General's Reports, would not call for any protest had it not been quoted and accepted by Hilton Fagge, who founded upon this supposed prevalence, taken together with the known rarity of gout in Scotland, the wrong conclusion that gout and diabetes are in nowise related.

All observers are agreed that Jews are specially liable to become diabetic. There is some reason to believe that modern life is in itself a cause of diabetes. In his admirable statistics for the city of Paris,[1] Dr. Bertillon has demonstrated that the mortality from diabetes is higher in all the wealthier *arrondissements;* while in India and Ceylon it is among the educated and commercial and not among the labouring classes that the numerous cases of diabetes occur. A person belonging to the richer classes in towns usually eats too much; spends a great part of his life indoors; takes too little bodily exercise, and overtaxes his nervous system in the pursuit of knowledge, business or pleasure. Although there are many exceptions to such a description, it is a perfectly accurate account of large classes, and especially of the well-to-do Jew, who raises himself

[1] TABLE showing the Average Annual Mortality from Diabetes per 100,000 of Population for each *Arrondissement* of Paris during three quinquennial periods.

Name.	1865-69.	1881-85.	1886-90.	Remarks.
Louvre	3	5	11	Very well-to-do.
Bourse	2	7	10	"
Temple	2	12	16	Well-to-do.
Hôtel-de-Ville	3	8	13	"
Panthéon	2	6	10	"
Luxembourg	3	11	15	Rich.
Palais-Bourbon	4	9	14	"
Elysée	4	12	16	Very rich.
Opéra	5	13	20	Rich.
St. Laurent	4	10	12	Very well-to-do.
Popincourt	2	6	11	Poor.
Reuilly	2	8	11	"
Gobelins	1	8	9	Very poor.
Observatoire	2	7	9	Poor.
Vaugirard	1	6	14	"
Passy	2	12	18	Rich.
Batignolles	3	8	14	Well-to-do.
Montmartre	1	7	9	Very poor.
Buttes-Chaumont	1	4	7	"
Ménilmontant	2	5	8	"
All Paris	4	8	13	
Males	3	10	15	
Females	2	6	10	

easily by his superior mental ability to a comfortable social position, and notoriously avoids all kinds of bodily exercises.

The excessive use of sugar as food is very generally believed to be a cause of this disease, although it is not easy to see how it acts. The fondness of Jews for sweet things has been suggested as one explanation of the frequency with which they become diabetic; and local practitioners in Persia, India and Ceylon are accustomed, rightly or wrongly, to attribute the disease to abuse of sweetmeats and sweet sherbets. Even the exclusive employment of vegetable food has been blamed for the same reason; and Charcot stated that temporary glycosuria is often observed among the novices at La Trappe. But in Mauritius and British Guiana—countries in which the people are chiefly employed in the manufacture of sugar, in Africa where the sugar-cane and other sweet fruits and vegetables form a large part of the diet of the natives, and in Ireland where the food of the peasantry is mainly vegetable, the disease is relatively rare. As little valuable are the opinions that beer-drinking and cider-drinking are efficient causes: the deaths from diabetes in the cider-drinking counties of England are below the average, while the kingdom of Prussia, which probably stands at the head of the beer-consuming countries of Europe, has but a low diabetic mortality.

No *age* is exempt, but diabetes occurs most commonly in the fifth decade of life, being rarer at its two extremes. Cases have been observed in infants a few weeks old, and in aged persons over eighty. The greatest mortality is between the ages of fifty-five and seventy-five—that is some years later than the period at which it most frequently supervenes, for elderly diabetics are able to sustain the disease for many years.

The different incidence of the disease in the two *sexes* is very marked in tables[1] which include deaths at all ages, being in the proportion of 3 males to 2 females; but when the cases are analysed so as to admit of comparison at the several periods of life, this difference is seen to be much less in childhood and extreme old age when the figures are more nearly equal.

Heredity is an influence too well attested in certain instances to be doubted; thus in one example diabetes occurred in eight members of one family extending over three generations.

It is also not uncommon to meet with a history of *insanity*, *phthisis*, or *gout* among the relatives of diabetics.

[1] England and Wales.

All Ages.	Under 1 year.	1 year.	2 years.	3 years.	4 years.	Total under 5 years.	5 years.	10 years.	15 years.	20 years.	25 years.	35 years.	45 years.	55 years.	65 years.	75 years.	85 and upwards.
Males, 1142 .	3	4	2	1	1	11	3	30	44	47	116	130	192	262	233	67	7
Females, 869 .	...	..	..	1	3	4	7	28	34	48	104	84	125	198	177	57	3
Total, 2011 .	3	4	2	2	4	15	10	58	78	95	220	214	317	460	410	124	10

Obesity is supposed by many persons to favour the occurrence of diabetes: undoubtedly the coincidence is common enough, but the opinion of Kisch, that it directly arises from the muscles becoming so infiltrated with fat that they are thereby unable to do their share in the destruction of sugar, is open to doubt. It is more probable that obesity is the outcome of similar general conditions. Where the supply of carbohydrates is in excess of the demands of the tissues or of their consuming capacity, it is stored as fat; and if we regard obesity as an indication of such an excess, it is not difficult to suppose a further stage at which, the limits of fat-storage being reached, the surplus sugar circulating in the blood passes out of the body by the kidneys, and glycosuria, transient or permanent, ensues. The excessive supply of carbohydrates, whether in the form of sugar, vegetable food, or beer, does no harm in the absence of the essential causes of diabetes; the excess is stored up as fat, or may even appear in the urine as a transient glycosuria. But when an excess of glucose, greatly beyond the power of the organism to utilise, is constantly poured into the circulation, we have the condition set up that we call diabetes; this must appear all the more rapidly where the carbohydrate supply is already very great.

The exact relation of *gout* to diabetes is an open question. No one who has an adequate clinical knowledge of the two diseases doubts the frequency of their association. It is possible that an acute attack of gout may be the direct cause of the onset of diabetes; but it is not clear that the gouty diathesis in itself disposes to diabetes except in so far as it is evidence of the pathological results of modern civilisation in certain individuals. There are many gouty families and more gouty individuals in whom the diathesis has its most typical expression without resulting in the production of diabetes.

The list of the *immediate causes* of diabetes is a very long one, but in most instances the relationship reposes on too well established a basis of clinical evidence to be rejected.

In the first place comes the long catalogue of injuries, such as railway accidents, severe contusions of the head, spine or abdomen, violent muscular strains, and lightning stroke, which are so frequently followed after a longer or shorter interval by diabetes. In all these cases the disease is probably produced by some derangement of the innervation of the liver.

Closely allied, therefore, to these are the rarer examples in which the disease is actually associated with some coarse lesion of the medulla; as, for example, where this structure is implicated in a new growth, or invaded by sclerosis (tabes dorsalis, insular sclerosis); or where it occurs in the course of insanity, epilepsy, or Graves' disease.

Possibly we ought to group under nervous influences those cases which have been attributed to climacteric disturbance, excessive sexual indulgence, and the action of certain poisons; for example, chloroform (Fort), bromide of potassium (Weber).

In the second place, but in quite a different category, come the

cases which follow the various acute infectious diseases, among which numerous well-attested examples have been recorded as sequels of enteric fever, diphtheria, tonsillitis (?), influenza, acute rheumatism, malaria, and syphilis. Insufficient attention has been paid to the possibility of diabetes having in some cases a microbic origin. Charrin and Carnot have proved that it is possible to render a dog diabetic by injecting infective fluids (bacillus coli, bacillus pyocyaneus and streptococci) into the pancreatic duct; and it is probable that the acute infective diseases diminish the resistance of the pancreas to the invasion of those organisms which occur so abundantly in the intestine.

There is no evidence at present of any specific microbic cause for diabetes; but in connection with this aspect of the subject I may refer to the many examples of diabetes occurring in husband and wife, which have led some writers to speculate on the possibility of its transmission by contagion (Dréfous, Gaucher, Labbé, Letulle, Schmitz, Rendu).

We may group with the above those cases which have been recorded in connection with pancreatic cancer, cysts, and calculi.

In a third class must be placed the cases which have their apparent starting-point in pregnancy and parturition, as their explanation remains obscure.

Beside the above, are the cases associated with abscess of the liver, malignant disease in the abdomen not involving the pancreas, exposure to severe cold, and the ingestion of cold food (ices) or iced drinks.

Pathogenesis.—Diabetes has been already defined as persistent glycosuria. By this we mean the constant presence of glucose in the urine in quantity sufficient for discovery by ordinary tests. Glucose ($C_6H_{12}O_6$), now regarded as a ketone of a hexatomic alcohol, exists in two modifications, dextrose and lævulose; but it usually occurs in the urine only as the former, lævulose taken as food being excreted as dextrose. The sucroses $(C_{12}H_{22}O_{11})_7$—cane-sugar, maltose, and lactose (the last two are physiological products, and lactose is occasionally met with in the urine of nursing women)—are held to be constituted by the union of two molecules of glucose with loss of H_2O, and are called disaccharids. Starch, dextrin, and glycogen are polysaccharids formed by the union and dehydration of several molecules of glucose. Under the influence of ferments or the action of dilute acids this process is reversed, and the complex molecule of starch is split up and hydrated to form maltose or glucose, as in ordinary digestion.

Traces of glucose are met with constantly in normal blood (0·5 per mille), and corresponding traces can be detected in the urine by concentrating a large bulk; but it is not with these small quantities that we are concerned in diabetes: in this disease the blood contains excess of sugar (as much as 0·5 per cent) and the urine is loaded with it.

The first light thrown upon the pathology of glycosuria was the discovery by Cl. Bernard (1849) that the liver after death contains sugar; and his inference that the liver is a sugar-forming gland has withstood all the criticism to which it has been subjected. Bernard's researches did not

stop here; he found that the liver stored carbohydrates in the form of glycogen, and he believed that this substance was converted into sugar under the influence of a diastatic ferment contained in the blood: moreover, by means of puncture of the floor of the fourth ventricle he succeeded in determining such an excessive production of sugar that the excess passed into the urine. According to Bernard, carbohydrates are absorbed from the intestine as sugar, and are carried by the portal vein to the liver, where they are stored as glycogen. This stored material is given out gradually as it is required to meet the needs of the economy, being converted into glucose and carried by the hepatic vein into the general circulation.

Bernard's views have been opposed with great ability and pertinacity by Dr. Pavy, but, in spite of much effective criticism of his methods, the result has been to leave his conclusions substantially intact. Bernard's doctrines have found followers able to demonstrate the truth of his statements by more exact means; and at the present time the only important modification towards which opinion seems to tend is the rejection of the agency of a glycogenic ferment in the liver in favour of the conception of glycogenesis as a direct and normal function of the liver-cells.

Dr. Pavy thinks that glycogen may be converted directly into fat; and there is good ground for his inquiry into the fate of the great excess of glycogen found in the livers of vegetable-feeders, for the blood of these animals contains no more sugar than does that of flesh-eaters, although their store of glycogen is so much greater. Pavy has made an interesting observation on the presence of fat in the intestinal villi of a long-starved rabbit, which suggests that starch may be converted into fat by the epithelium of the intestinal villi; and he thinks that it shares this function with the liver-cells.

That carbohydrates are not the only possible sources of sugar is proved by the presence of glucose in the blood of animals fed on lean meat only; and our clinical experience of diabetes proves but too frequently how large a quantity of sugar may be excreted by patients whose diet contains very little carbohydrates. Seegen believes that sugar is formed from peptone, and on insufficient grounds denies that glycogen is its principal source; and Pavy has shown that sugar may be artificially produced from proteid matter. It is certain that sugar can be formed from the proteid of the food and from the tissues; and the question whether it is derived from the proteid molecule by splitting off, or whether the molecule is first completely broken down and then built up again as carbohydrate and urea, is one which need not detain us; the latter view has, perhaps, the greater weight of authority. The seat of this process, or the agent which determines it in the body, is not definitely known; but it is assumed that it takes place in the liver, and that the liver-cells effect the conversion. But what becomes of the sugar poured into the circulation?

Under the influence of Liebig's teaching it used to be believed that glucose was burnt off in the lungs and excreted as water and CO_2, the

process subserving the maintenance of the body temperature. But of late years the seat of this destructive process has been shifted to the muscles, which are said to store up glucose in the form of glycogen, to be utilised when in action. This view is supported by the disappearance of glycogen from muscles which have been tetanised by strychnine poisoning; and by the destruction of sugar in the tissues proved to take place by comparison of the amount contained in arterial and venous blood respectively.

While Claude Bernard and those who have followed him in the same line of research have done much as pioneers, it cannot be said that they have succeeded in indicating the direction in which the solution of the problem of the pathogenesis of diabetes is to be found. The most careful research has failed to establish that structural disease in the floor of the fourth ventricle or any part of the nervous system or in the liver is even a common occurrence in diabetes. On the contrary, in the early stages of the disease these organs appear perfectly healthy; so that if they be primarily concerned, diabetes can only be regarded as a *functional disease of the nervous system.*

Besides the "diabetic puncture" the following experimental lesions of the nervous system are said to be followed by glycosuria:—

(i.) Injury to the vermiform process of the cerebellum: (ii.) Section of the spinal cord at various levels: (iii.) Section of the anterior cervical nerve-roots: (iv.) Section of the posterior cervical nerve-roots: (v.) Artificial neuritis of the first pair of dorsal nerves: (vi.) Destruction of the superior and inferior cervical sympathetic ganglia, of the first thoracic, and of the abdominal ganglia: (vii.) Section of or ligaturing the splanchnic nerves: (viii.) Irritation of the right vagus: (ix.) Section and stimulation of the central end of an ordinary sensorimotor nerve such as the sciatic

Under the influence of these various lesions the glycogenic function of the liver is enormously increased, the quantity of sugar poured into the circulation far exceeds the consuming and storing powers of the tissues, and glycosuria is the inevitable consequence. But the effect of these lesions is, as a rule, transitory, resembling in this respect the majority of cases of traumatic glycosuria in man.

It is probable that some poisons produce glycosuria in the same way, while others are merely converted into reducing substances (for example, glycuronic acid), which are excreted in the urine and are there mistaken for sugar.

In 1877 Lancereaux drew the attention of the French Academy of Medicine to the changes in the *pancreas* which, he believed, were constantly present in severe cases of diabetes; and within the last few years the importance of this communication has been demonstrated by the experimental production of diabetes in dogs by extirpation of this gland.

Von Mering and Minkowski have found that after this operation the animals suffer from glycosuria, polyuria, great thirst, hunger, and rapid emaciation; in fact, all the classical symptoms of diabetes. On the other

hand, mere obstruction of the pancreatic duct—by ligature or by injection with paraffin (Hédon) or asphaltum (Thiroloix)—is not followed by any of these symptoms; nor do they occur if a small part of the gland be left, even although the duct be removed. These statements have been subjected to most severe criticism, and the experiments have been repeated in numberless instances, with the result that their accuracy has been established. It was at first suggested that the operation injured the solar plexus and so set up nervous diabetes; but apart from the statement of the operators that no such injury had been inflicted, the absence of the symptoms when a part of the gland was left contradicted this view. But the most convincing proof that such an injury cannot be the explanation is afforded by the experiment of grafting a portion of the extirpated pancreas outside the abdominal cavity in the muscles of the external walls; this operation has been successfully performed by several independent investigators (Minkowski, Hédon, Thiroloix) with the result of preventing the occurrence of diabetes just as if the part of the gland had been left in place.

Inspection of the bodies of diabetic patients has proved that alterations of the pancreas are very commonly present; their nature and frequency will be considered hereafter. It is enough now to know that they exist, and that experiment and morbid anatomy are not here so strikingly at variance as in other theories of diabetes.

The means by which the presence or absence of the pancreas influences the production of glycosuria needs further explanation. Lépine has afforded the best solution of this difficulty by the supposition that the pancreas secretes a sugar-destroying ferment, which passes directly into the chyle and blood. He asserts that these fluids in the normal state possess such an action to a powerful extent, and that this action disappears when the pancreas has been extirpated; but Lépine's statements are denied by Arthus, Gaglio, and Sansoni: moreover, no one has yet succeeded in obtaining such a ferment from the pancreas, and pancreatic extracts, however administered, have failed to control glycosuria. The pancreatic hypothesis of diabetes is undoubtedly a great advance towards a satisfactory pathology of the disease; but as in certain cases the gland appears to be quite normal, it does not cover the whole ground, and it leaves room for the old view of its nervous origin, at least in respect of a certain proportion of cases. Kaufmann has suggested a modification of Lépine's hypothesis, namely, that the secretion controls the sugar-forming function of the liver; he believes he has proved by analysis of arterial and venous blood in healthy and diabetic animals that the tissues of the latter consume quite the normal proportion of sugar: it was formerly believed that the gaseous exchanges of diabetes gave evidence of diminished oxidation, but the results of later experiments contradict this opinion (Leo, Weintraud, Laves), he therefore regards the theory of insufficient destruction of sugar as untenable. Von Noorden, on the other hand, denies the extra production of sugar, and finds the explanation of diabetes in diminished destruction in the tissues, loss of power to store carbohydrate as fat, and

incompetency of the glycogen reservoirs (liver, muscles). He assumes as a fact that every molecule of albumin breaks up into carbohydrate and urea, and that for every gramme of urea 2·8 grammes of carbohydrate have been formed in the body; hence he concludes that it is impossible to show an excretion of sugar greater than may be derived in this fashion.

He disregards the various authors (Voit, Leo, and others) who have shown that there is no decrease of CO_2 production in diabetes, and attaches great weight to the single observation of Weintraud and Laves, that the addition of small quantities of carbohydrate to the dietary of diabetics raises their respiratory quotient (the proportion of CO_2 to O) much less than in health. Finally, he discredits the results of those observers who have found that the sugar is no greater in arterial than in venous blood (Pavy, Beck, and Hoffmann, Abeles, Seegen); and although Chauveau and Kaufmann find that the arterial blood always contains more sugar, they do not help von Noorden, as the relation remains exactly the same in diabetic as in healthy animals.

Where the evidence is so conflicting, and the weight of authority so nicely balanced, we may well hesitate before ranging ourselves on one side of this controversy or on the other. Pavy believes the intestinal villi share with the liver the power to convert carbohydrates into fat, and he thinks this power is lost in diabetes; but the evidence of this power rests upon his own observation, hitherto unsupported, of the amount of fat demonstrable in the villi of a rabbit after a full meal of oats. Noel Paton thinks the oats contain enough oil to account for these appearances without assuming for them any less direct explanation. That the carbohydrates are converted into fat (Pavy) or actually consumed (Lépine) before they reach the liver, has not been proved; and either view is as difficult to accept as the ordinary one that all the carbohydrates are first stored in the liver, to be doled out thence in constant, regular measure to supply the needs of the body.

If the liver possess in health not only the power of sugar-formation, but some mechanism by which it adapts the amount manufactured and passed into the circulation to the current demands of the organism, and thus, whether the store of glycogen be great or small, keeps the percentage of sugar in the blood at a constant point, we have only to assume a disorder of this regulating function to explain the occurrence of diabetes.

In diabetes the liver continues to manufacture sugar constantly, utilising for the purpose its accumulated stores of glycogen, which it soon exhausts, and afterwards the carbohydrates and albumin of the food; apparently falling back upon the albumin when the former are deficient. This would constitute what is called over-production, although the amount may not exceed the value of the food ingested; but in health the quantity of food would probably be less, and the resulting glycogen would be stored up, no more than the required amount of sugar being manufactured to maintain the normal blood percentage. Thus the hyperglycæmia and glycosuria are due to the excess of sugar which the liver lets pass into the circulation, and not to failure on the part of the tissues to con-

sume a normal quantity. If there were such a diminished sugar-consumption there ought to be a demonstrable fall in the CO_2 eliminated; but, as has been already stated, the CO_2 output is normal, and the amount of CO_2 in the blood is not less than in health (Leo).

Recent physiological researches have rendered it probable that sugar-formation is a function of the liver-cells, under the control of certain undetermined excito-secretory nerves which, according to Morat and Dufour, are distinct from the vaso-motor nerves. It is quite conceivable that this nervous influence may be liberated by various injuries to the nervous system, or by the action of poisons; and the extirpation of the pancreas may also act in some unknown way upon this function, but we are not in a position to do more than speculate upon these possible explanations.

The glycosuria which follows the administration of phloridzin ($C_{21}H_{24}O_{10} \times H_2O$), a glucoside obtained from the bark of apple, pear, cherry, and plum trees, is said by Minkowski to be genuine, and not due to the presence of its decomposition product, phlorosin, which has the formula of sugar, reduces cupric oxide and undergoes fermentation; as the administration of one gramme of phloridzin was followed by the excretion of 97 grammes of glucose, this must be admitted. It is believed that the drug acts by altering the renal epithelium so that it no longer holds back sugar, and this view, as von Noorden points out, reminds us of the old theory of the renal origin of diabetes, and suggests that it possibly plays some part in the pathogenesis of certain forms of the disease.

Cantani's hypothesis that the sugar of diabetes is a paraglucose incapable of subserving the needs of the body, and therefore excreted unchanged, has received no support from any recent chemical or physiological discoveries. Nor can we say any more in favour of the doctrine propounded by Latham, that diabetes is due to imperfect oxidation of the muscle-albumin and the formation of sugar by the condensation of six molecules of methyl aldehyde. Dr. Pavy's latest researches have left his improved hypothesis of vaso-motor paralysis of the vessels in the splanchnic area exactly where it was; its place in the nervous theory is now occupied by the conception of an excito-secretory nerve which controls the sugar-forming function of the liver, and which, as we have already seen, may be affected by various stimulating or depressing agencies.

Morbid anatomy.—The bodies of persons dying from diabetes present as many departures from the healthy standard as might be expected in the case of a disease in which nutrition is so gravely impaired; but most of these changes are to be regarded as simple failure in the reparative processes of the tissues.

External appearances.—There is generally wasting and sometimes extreme emaciation; but in other cases a well-developed layer of subcutaneous fat may be present. The skin is thin and harsh, the hair scanty and dry, the teeth very defective, and there are frequently traces of rashes, or scars of boils and carbuncles.

Brain.—This organ presents no constant lesion, but was normal only in eight out of thirty-one cases under my own observation. The most common change is congestion and œdema, with thickening of the membranes. It is less often anæmic; sometimes the convolutions are wasted and the sulci widened. The brain tissue may contain cavities which have been aptly compared to those met with in Gruyère cheese; they vary in size from a pin's head to a horse-bean, and seem to depend upon local atrophy from failure of nutrition. The lateral ventricles and the iter have been found dilated without any obvious mechanical cause. The choroid plexuses may be congested or thickened, or may contain cysts.

Of much greater importance are the tumours of the fourth ventricle and medulla, of which ten cases have been recorded; in many of these there can be no reasonable doubt that the diabetes depended directly upon the growth. In the same situation also have been observed examples of softening, of sclerosis, of alterations of colour and congestion, and of the presence of corpora amylacea and colloid masses. The softening or sclerosis in this position may undoubtedly cause diabetes, but the other lesions are of more doubtful value. The enlargement of the perivascular spaces, to which Dr. Dickinson drew attention, is only a consequence of the failure of brain nutrition, as in the case of the cysts already described.

Extensive hæmorrhage into the brain occurs rarely. Glycogen is present in large quantities in the medulla oblongata and in the sheaths of the vessels of the cortex.

In the microscopical examination of numerous specimens by myself no special or characteristic structural changes could be detected in any part of the brain. No minute hæmorrhages were found, and the cysts already mentioned showed no trace of hæmatoidin-staining. In only one instance did the capillaries of the vagus nucleus seem to be abnormally numerous and full of blood.

Spinal cord.—A certain number of cases of diabetes undoubtedly occur from the extension of disease from the cord into the medulla, and when this happens changes characteristic of locomotor ataxia, insular sclerosis, or like lesions, are present. We do not possess abundant materials for making very positive statements as to the general condition of the cord, for examination of it has often been omitted. The recorded facts point to the frequency of secondary nutritive changes; for example, dilatation of the central canal, enlargement of the perivascular sheaths, and localised softening. Some recent observations by Sandmeyer and by Williamson have indicated that slight atrophic changes may occur in the posterior columns, chiefly marked by their failure to react normally to staining agents, although no symptoms of tabes dorsalis may have been present during life. The only case of tumour of the cord followed by diabetes is one of myxoma of the dura mater recorded by Dr. Shingleton Smith. Glycogen is present in large quantities in the spinal membranes and sheaths of the vessels.

Cerebro-spinal nerves.—Three examples have been recorded of diabetes caused by tumours growing from the right vagus; though in one instance (that of Frerichs) the growth encroached upon the floor of the fourth ventricle. Lubimoff has also described a case of diabetes in which atrophy and pigmentation of the inferior ganglion of the vagus were present.

The peripheral nerves may be the seat of interstitial inflammation, which is characterised by great increase of connective tissue, with secondary destruction of the axis cylinders; and this change may cause well-marked clinical symptoms. The nerves of the lower extremities are chiefly affected, but any part may be attacked.

Sympathetic nerves and ganglia.—Changes in these structures early attracted the attention of students of the pathology of diabetes, as they occur with some frequency. Duncan, in 1818, found the sympathetic trunk in the abdomen three times as thick as normal. Percy, in 1842, described the semilunar ganglia, the splanchnic nerves and vagi, as thickened and of cartilaginous hardness. Lubimoff found sclerosis of the sympathetic ganglia and atrophy of their nerve-cells. Cavazzani has described atrophy of the cœliac plexus. The semilunar ganglia in my cases have been four times enlarged, once atrophied, and once embedded in a mass of fibrous tissue; but, as a rule, they were normal, and the histological changes the same as are met with in many other maladies, although these ganglia are never so greatly enlarged as in diabetes.

In spite, therefore, of the extent of these changes, they are not to be regarded as the cause of the disease.

Heart.—The myocardium is often pale and soft, more rarely hypertrophied or distinctly fatty. Pericarditis occurs occasionally and endocarditis sometimes, while coincident valvular disease may be present. Advanced fatty degeneration of the muscular fibres is the characteristic change in old-standing cases of diabetes. Glycogen granules may be found between the muscular bundles.

Blood.—The appearance of the blood is generally normal; but at times it is loaded with fat, which, after the blood is shed, floats on its surface in a cream-like layer. Under the microscope this fat is seen to be present as a very fine emulsion, but the granules run together after death to form droplets, and thus give rise to the appearance of fat embolism in the capillaries: this, however, is but a post-mortem change. The red corpuscles are sometimes broken down into a granular material, and they are generally reduced in number. There may be an abnormal abundance of megalocytes or of large mononucleated leucocytes.

Chemically the blood contains, as a rule, more sugar than it does in health, and the proportion may be as high as 4 per mille. The alkalinity of the blood-serum is reduced in consequence of the presence of acids of doubtful identity, β-oxybutyric acid and diacetic acid being the most probable. The presence of acetone, which may be due to the splitting up of diacetic acid into acetone and CO_2, has been observed, but is not constant even after death from coma.

Lungs.—Secondary changes in these organs are very common, the most usual being congestion and œdema. Next in frequency comes phthisis, usually but, as proved by the case recorded by Roque Dévic, not invariably tubercular. Lobar pneumonia occurs rarely, but is very acute and fatal; acute broncho-pneumonia is sometimes met with. Small foci of softening, abscesses, hæmorrhagic infarcts, and gangrene may be found. Pleurisy and empyema occur sometimes. Fat embolisms of the pulmonary capillaries have been described, but these are really post-mortem changes in the fatty blood. Hyaline fibroid thickening of the vessels occurs as part of the tissue changes of chronic inflammation.

Liver.—This organ is generally enlarged, weighing from 60 to 80 ounces; less commonly it is small, pale and soft. It is sometimes fatty, often congested, and its consistence may be abnormally firm. It frequently presents a certain amount of interstitial hepatitis, and occasionally it may be distinctly cirrhotic. The new growth commences in both the hepatic and portal areas. This form of cirrhosis is sometimes associated with bronzing of the skin (Hanot and Schachmann). Abscess of the liver is occasionally met with in cases of diabetes, and then is probably a causal lesion. Dr. Dickinson has described thrombosis of branches of the portal vein, and angiomas formed of dilated capillaries near the radicles of the hepatic vein. According to Weyl and Apt the diabetic liver does not contain excess of fat, and absence of this substance from the hepatic cells has been noticed by Beale and Frerichs. Quincke thinks that iron is in excess, but Zaleski estimates it at 0·685 per mille, and points out that we have no data to enable us to say whether this amount is greater than normal or not.

Spleen.—This organ is commonly said to be small, pale, and soft: less often it is enlarged and congested; sometimes it contains tubercle; glycogen has been found here as elsewhere. Hyaline degeneration of the vessels has been described.

Pancreas.—Great interest attaches to the alterations in this organ, since the important researches of Minkowski have shown that its complete destruction in dogs is followed constantly by all the symptoms of diabetes; and many papers have been published on the subject (Baumel, Churton, Vaughan Harley, Williamson, G. Hoppe Seyler, Hansemann, Fleiner, and others). Unfortunately we have still a good deal to learn about the significance of slighter changes in its structure, and it is possible that, while appearing normal to the naked eye, it may be extensively diseased. Lancereaux thought that pancreatic disease was invariably associated with the clinical type which he called diabète maigre; and this association is undoubtedly the rule although, since attention has been drawn to the matter, many exceptions have been recorded, and fat diabetics have been shown to present well-marked pancreatic atrophy (Baúmel, Lépine, G. Hoppe Seyler, Williamson). As to the relative frequency of these changes my notes of 27 consecutive cases give the following results:—

Pancreas atrophied	13
,, large and hard	5
,, large and soft	1
,, large	1
,, congested (mottled)	1
,, normal	6
	27

In two cases reported by Fleiner attacks of pancreatic colic had preceded for years the onset of diabetes, which, in fact, did not supervene until cirrhosis had destroyed the entire gland. Several cases of cancer of the pancreas associated with all the symptoms of diabetes have been published. Other cases are on record of diabetes with cystic disease of the pancreas and with multiple pancreatic abscesses.

In the morbid specimens examined the lesions described in my Bradshaw lecture (1890) have always been found, namely, varying degrees of interstitial inflammation with formation of large areas of connective tissue and new ducts; hyaline changes in the epithelium are constant, but probably are not pathological, as they may be found in glands which are otherwise healthy. In the earlier stages of the process the gland becomes swollen and infiltrated with small round cells. Besides this cirrhotic atrophy the gland may undergo fatty degeneration, which leads to complete transformation of its entire substance into fat. It has been suggested that the changes in the pancreas are secondary to disease of the cœliac plexus, but Lustig and Peiper have shown that extirpation of this plexus is not followed by atrophy of the pancreas.

Stomach.—Although our data are by no means complete on this part of our subject, this viscus is seldom found to be normal. It is often dilated, and shows evidence of chronic gastritis; the mucosa may be congested or contain hæmorrhages, and is not uncommonly thickened.

Intestines.—These share in the congestion and catarrh which are found in the stomach: hæmorrhages may be present in the duodenum. The large intestine is generally filled with hardened fæces, and occasionally shows dysenteric inflammation or desquamation of its epithelial lining.

Kidneys.—Although changes in these organs are undoubtedly secondary and variable in kind and degree, they are always present. They generally consist in enlargement and slight fatty degeneration; less commonly congestion is observed. Sometimes the cortex is thinned and the organ contracted. Tubercle, lardaceous disease, and even gangrene have been found. But the only distinctive lesion is the hyaline transformation of the epithelium of Henle's tubes first described by Armanni, and named after him. It is not constantly present, and its etiology is still obscure. Glycogen may be found in the renal epithelium, as elsewhere, and marked fatty degeneration may be present. Not uncommonly the kidneys exhibit all the characteristic histological appearances of chronic diffuse nephritis.

Bladder.—This viscus is usually normal, but may be dilated and hypertrophied, while its mucous lining may be the seat of hæmorrhages or catarrhal changes.

Summary.—Diabetes is a disease which has so profound an influence upon the general nutrition of the body, that it tends to produce structural alterations in the various organs, which are for the most part of a secondary and degenerative character. The exceptions are (i.) the tumours and growths in or near the medulla oblongata and the vagi nerves; (ii.) a few instances of primary liver disease; (iii.) cirrhosis and other destructive changes of the pancreas. The one important addition to the morbid anatomy of diabetes which the last few years have yielded is undoubtedly the lesions of the pancreas; and we are justified in regarding these changes, when present, as the cause of the symptoms in the same sense as granular kidney is the cause of the symptoms of chronic Bright's disease.

Symptoms and course.—Diabetes manifests itself in two principal clinical forms—(*a*) acute, and (*b*) chronic, which differ in the intensity of the essential symptoms: these are glycosuria, polyuria, thirst, and wasting—symptoms which are always excessive in the acute and more moderate in the chronic form. Acute diabetes usually occurs in persons under forty years of age, and not uncommonly in children or young adults. The patient complains of weakness, of thirst, and of passing an excessive quantity of water. The frequency of micturition interferes greatly with sleep, and the want of rest is one of the chief causes of the great constitutional depression. In spite of an appetite which may be voracious the body-weight diminishes rapidly; the face is often flushed, the skin dry, the hair rough, the lips parched, the tongue red and sticky, or covered with a black fur, the secretions of the mouth diminished, and the bowels confined. There is often a persistent nauseous sweet taste in the mouth, the breath may have a sweet odour; the muscular strength is much impaired; sexual appetite is usually lost, and the mind is depressed. The quantity of urine varies from five to fifteen pints or more, and contains from 5 to 10 or 12 per cent of sugar.

Chronic diabetes, on the other hand, occurs as a rule in elderly people of both sexes, and often in those who are or have been decidedly obese. Such patients complain of weakness, of frequent micturition leading to disturbed nights and of some loss of flesh; sexual desire is generally absent; the mind is often depressed, and the digestive organs are disturbed. The quantity of urine may vary from three to six pints, and the sugar from 3 to 10 per cent.

There is nothing constant in the external appearance of a diabetic patient; although the peculiar flush which, when present, is not limited to the malar eminences but resembles a deep blush extending up to the roots of the hair, may suggest to an experienced observer the true nature of the case. The odour of the breath is also significant. The nutrition of the skin and its appendages suffers constantly; so that the epidermis becomes dry and rough, the nails brittle, and the hair thin and dry. The temperature of the body is usually subnormal, but an increase has

been observed at the onset of some acute cases reaching as high as 103° F. (39·4 C.)

As already mentioned there may be some mental depression and irritability of temper; vision is frequently impaired, the other senses less commonly so; common sensibility to touch and pain, and sensations of heat and cold, remain normal; neuralgic affections are frequently observed, and occasionally there may be paralysis or ataxy from peripheral neuritis. The knee-jerks are sometimes diminished or lost, but are generally normal. In women menstruation is as a rule deficient or absent. The appetite is usually good and digestion easy; but the bowels are almost always confined, though in some cases there is a marked tendency to diarrhœa. The teeth are very often decayed or falling out from atrophy of the gums. The stools have a peculiarly fœtid odour. The cardiac impulse is usually in the normal position, and in advanced cases it is diffused and weak. The pulse, which at first shows a tracing of high pressure, becomes, in the later stages, small and feeble, but its rate is not increased except in consequence of some complication. A rapid pulse is one of the early signs of the onset of coma.

The blood contains excess of sugar, and often a marked increase of fat; its alkalinity may be reduced 50 per cent, but its microscopic appearances are generally normal. In advanced cases there may be a great reduction of the red blood corpuscles (50 per cent), and it has been asserted that they do not stain normally with eosin (Bremer), but this change is certainly not constant. The serum is poisonous to rabbits (Roque Dévic). Sugar may be present in the sweat, tears, and saliva. The body-weight often undergoes rapid reduction before the institution of proper treatment, but after this has commenced it may vary from day to day within three or four pounds, although there may be no constant progress in gain or loss.

The persistent presence of sugar in the urine is the cardinal symptom of the disease, and we will therefore inquire whether glycosuria ever occurs apart from diabetes. Traces of sugar, as we have seen, may be demonstrated in normal urine; but in the urine of non-diabetic patients it is rare to meet with a quantity sufficient for demonstration by the ordinary tests. It has been stated that the urine of healthy breast-fed infants contains a substance which reduces cupric salts, but does not ferment. So-called "alimentary glycosuria" sometimes occurs in persons who have taken much sugar in food or drink, and it remains an open question whether such an occurrence indicates a tendency to diabetes. I have recorded an instance of persistent glycosuria in a man who appeared quite healthy, and at the present time, eight years after I first found sugar in his urine, he is in good health, although Fehling's solution is still reduced by his urine to a degree indicative of 1·2° per cent of sugar.

Temporary glycosuria occurs in many surgical conditions, after injuries, in some nervous diseases, in gout and other general maladies.

However, under the heading of the various tests for sugar, certain fallacies will be pointed out which in the past have led to many erroneous statements of the presence of sugar in the urine.

Urine.—The urine is generally greatly increased in quantity, but varies within wide limits; when diarrhœa is persistent it may be normal or even less than normal in amount. The specific gravity of the urine is usually high, varying from 1025 to 1050; but a low density does not exclude sugar, nor a high density prove its presence. In some of my cases it has been as low as 1013, while phosphatic urine may be as high as 1040. The colour in typical specimens is pale greenish yellow, but it varies through all shades of yellow up to deep amber; the liquid is generally clear, but may be turbid from lithates, muco-pus, or torulæ. It does not tend to decompose so readily as normal urine, but affords a favourable medium for the growth of the yeast plant. Its reaction is, almost invariably, strongly acid; and it deposits very commonly a considerable amount of uric acid crystals. In women some vaginitis is often present, so that the urine contains pus and epithelium. The normal constituents are increased; there is an absolute excess of water, chlorides, sulphates, kreatinin, ammonia, phosphates and urea. The phosphates and urea usually bear the proportion to each other of one to ten (Butel); and the urea to sugar of one to two (Harrison and Slater).

When carbohydrates are added to the diet the amount of sugar increases, but the urea diminishes almost *pari passu*. The loss of lime in combination as phosphate is very great, and the amount even approximates to that observed in mollities ossium. Some authors recognise a phosphatic diabetes, and cases occur in which phosphaturia precedes or alternates with glycosuria. The amount of ammonia eliminated is very large (Hallervorden), and this is attributed by Stadelmann to the excess of acid in the blood, which disturbs the normal mechanism for fixing ammonia; he therefore regards it as an index to the blood condition and a premonitory sign of diabetic coma.

The *amount of sugar* may vary up to 12 per cent. In some rare cases *air* has been found mixed with urine on its passage from the bladder, and it has been suggested that this is due to fermentative decomposition of sugar in the bladder with formation of CO_2, but the presence of alcohol has not been demonstrated. *Glycogen* is said to be constantly present, and *lævulose* has been met with. In some cases the sugar may disappear for a time and then return. *Albumin*, if present, may be due to an admixture of discharges, as of vaginitis and vesical catarrh, or to the presence of chronic nephritis. It is less common in young subjects; it is generally but not constantly present in the urine of cases of death from coma. Indican and skatoxylsulphuric acid may be present in excess; acetone, aceto-acetic acid, β-oxybutyric acid, and β-crotonic acid—all chemically related substances—have been found, and have excited much interest, as each or all of them has been supposed to be the poison which causes Küssmaul's coma; and some of them give the well-known Burgundy-red coloration with ferric chloride. This reaction is also

given by formic acid which is alleged to be sometimes present. Oxalic and hippuric acids frequently occur.

Tests for sugar.—Fermentation was the earliest method devised to prove the presence of sugar in the urine, and it is still the best; but it is too tedious to be useful on all occasions, and some little care is needed to demonstrate the presence of small quantities. Under the influence of the torula cerevisiæ, or beer yeast, sugar splits up into ethyl alcohol and carbonic anhydride, the process taking place best at 35° C. (95° F.) The amount of sugar may be estimated by the diminished specific gravity of the urine, or by collecting the CO_2; one molecule of grape-sugar giving two molecules of CO_2. For this a volumetric analysis or special fermentation tube is necessary; but for qualitative testing an ordinary test-tube filled for two-thirds of its depth with mercury, and inverted over the same metal, will answer the purpose. A little tartaric acid should be added to the urine, and the yeast should be well washed. Fermentation is ordinarily effected by taking two separate specimens of the urine, adding yeast to one, and putting both for some hours in a warm place; after fermentation has taken place the densities of the two specimens are compared, and the amount of sugar is calculated according to Roberts' formula, by which for each degree of density lost is reckoned one grain of sugar per ounce. The method is very fairly exact.

The usual qualitative test is Fehling's solution. This is prepared by mixing equal quantities of two liquids, (*a*) and (*b*). (*a*) consists of pure sulphate of copper (34·639 grammes) dissolved in distilled water (500 c.c.); (*b*) consists of pure sodium potassium tartrate (173 grammes) dissolved in a solution of caustic soda (100 c.c.) of sp. gr. 1·34, and diluted with distilled water (to 500 c.c.) On mixing exactly equal quantities a clear deep blue liquid is found, of which 10 c.c. = 0·05 gramme of dry diabetic sugar. As the mixture is very liable to decompose, the liquids are better kept separate until required for use.

To determine the presence of sugar, about a drachm of the mixture, and an equal quantity or less of the urine, should be thoroughly boiled together. If sugar be present the cupric is reduced to cuprous oxide, and the yellow suboxide of copper is thrown down.

This test is unfortunately not absolutely certain, as the urine may contain other reducing substances, of which glycuronic acid, uric acid, kreatinin, and lactose are well known. The urine of persons taking salicylic acid or its salts, chloral, chloroform, lactic acid, or aldehyde may give this reaction. It is therefore often of great importance to employ a confirmatory test, especially where only a trace of sugar appears to be present. Many exist, but the best is that devised by von Jaksch, which depends upon the property of phenyl-hydrazin to form with grape-sugar a characteristic crystalline compound called phenyl-glucosazon. Two parts of hydrochlorate of phenyl-hydrazin ("twice as much of the salt as will lie on the point of the blade of a knife") and three of acetate of soda are placed in a test-tube containing three or four drachms of urine; if the salts do not dissolve when the fluid is

warmed a little water is added, and the tube placed for twenty to thirty minutes in boiling water. It is afterwards taken out and allowed to stand in cold water, when, if sugar be present, a yellow precipitate is formed at once which, under the microscope, consists of detached or radiating clusters of characteristic yellow needles. If albumin be present it is better to get rid of it by previous boiling and filtration.

Quantitative estimation is usually performed by Fehling's solution, of which 10 c.c. = 0·05 gramme of grape-sugar. To perform the estimation we require a burette graduated in cubic centimetres, a hundred-cubic-centimetre glass measure, a porcelain capsule, an iron tripod, and a spirit-lamp or Bunsen burner. Ten c.c. of Fehling's solution are measured off, diluted to 50 c.c. with distilled water, and placed to boil in the capsule. Ten c.c. of urine are now measured off, diluted, and well mixed with nine volumes of distilled water; the burette is then filled with the diluted urine to zero on the scale. When the Fehling solution begins to boil the diluted urine should be run into it drop by drop, the operator constantly stirring the mixture, and carefully watching the result until the blue colour entirely disappears, and the whole of the copper is reduced. The calculation is then readily made thus:—Suppose that 20 c.c. of diluted urine have been used to reduce 10 c.c. of Fehling, which is equal to 0·05 grape-sugar, the percentage is $\frac{0{\cdot}05 \times 100}{20} = \frac{5}{20} = 0{\cdot}25$; but as the urine was diluted ten times, $0{\cdot}25 \times 10 = 2{\cdot}5$ per cent. The method does not pretend to perfect accuracy, but it is quite sufficient for clinical purposes.

Grape-sugar deflects polarised light to the right hand, and upon this is based a method of estimation by means of a somewhat expensive instrument called a polarimeter. The operation needs some care and practice; but a number of experiments, made by Dr. J. W. Russell in my wards, showed that it may afford very uniform results, although these were constantly less than the figures obtained by fermentation and Fehling's method. This variation may have been due to the presence of other substances, such as lævulose and β-oxybutyric acid, which rotate light in the opposite direction, and consequently diminish the net amount.

Among other tests for sugar is Moore's, in which the urine is treated with liquor potassæ, and boiled; if sugar be present it is decomposed, and imparts an intense brown coloration to the fluid; but mucin and bile give the same reaction, and the alkali used must be free from lead, hence this test alone is not trustworthy nor is it very delicate. Böttger's test is performed by mixing a quantity of urine with an equal volume of concentrated solution of sodium carbonate, and adding a little basic bismuth nitrate. The mixture is then shaken and boiled. In the presence of sugar a black deposit is formed from the reduction of bismuth oxide. It is less sensitive than Fehling, and this precipitate occurs in the urine of persons taking rhubarb, and in the presence of pus, blood, or mucus. The test devised by Braun, and recommended by Johnson, is performed by adding to a drachm of urine ten drops of concentrated solution of picric acid, half a drachm of liq. potassæ and enough water to

make up two drachms. On boiling the mixture it becomes a deep reddish brown if sugar be present; but in the absence of sugar a certain amount of the same colour is often produced, so that Johnson holds that the fluid must become opaque; herein lies some doubt in many cases.

The indigo-carmine test first proposed by Mulder is neither sensitive nor accurate; the urine is treated with solution of sodium carbonate, and solution of indigo-carmine is added until the whole is freely coloured; on heating the colour changes to yellow in the presence of sugar, and becomes blue again on being shaken up with air.

Krismer's test is performed by taking equal parts of urine, liquor potassæ (B.P.), and solution of safranine (1 to 1000), and heating the mixture, which decolorises in the presence of sugar. The reaction is very delicate, but it has been used for too short a time to enable a very confident opinion to be expressed as to its trustworthiness.[1] It is said that some specimens of safranine act better than others.

Rubner's test for sugar is made by adding to 10 c.c. of urine an equal quantity of neutral acetate of lead, and filtering. To the filtrate ammonia is added drop by drop until a thick, curdy precipitate falls; this must be heated cautiously up to 80° C. (176° F.), when if grape-sugar is present it turns rosy red; on further heating it becomes coffee-brown. The reaction will indicate the presence of 0·25 per cent of sugar.

Acetone may be most readily detected in the urine by Le Nobel's method. A few drops of a weak solution of nitro-prusside of sodium (1 per cent), rendered alkaline by addition of ammonia, are added to the urine, which turns to an amethyst colour in the presence of acetone; but it requires some minutes to change. On boiling and acidulation this colour changes to greenish blue.

In Legal's test caustic potash takes the place of ammonia, and the resulting colour is red, which disappears on standing; on adding acetic acid a deep violet colour is produced.

Lieben's test, as modified by Ralfe, is performed by floating a drachm of urine upon a drachm of liq. potassæ containing 20 grains of iodide of potassium, and previously boiled in the test-tube. In the presence of acetone the phosphatic ring, which forms at the point of contact, turns yellow, or is studded with yellow points from the formation of crystals of iodoform; this reaction, however, has the disadvantage that it is produced by lactic acid and alcohol as well as by acetone.

The ferric chloride reaction is the name given to the Burgundy-red coloration assumed by urines on the addition of a few drops of liq. ferri perchloridi. It is probable that the cause of this reaction in diabetic urine is the presence of ethyl-diacetic acid, or diacetic acid as it is commonly called, an acid which readily breaks up to form acetone and carbonic anhydride; but many other more or less nearly allied substances also give this reaction, and the urine of patients taking carbolic or salicylic acid, antipyrin, thallin, and allied compounds which give a deep purple, may cause confusion. The characteristic colour is a deep

[1] Subsequent experience has confirmed this doubt.

rich Burgundy or claret colour. The reaction of diacetic acid may be distinguished by boiling another specimen of the urine before applying the test; as this acid is driven off by heat the reaction should fail. This reaction is not peculiar to diabetes, and is not of any very certain significance, though interesting and worth noting.

Prof. von Noorden attaches great importance to the presence of β-oxybutyric acid in the urine, for he regards this substance as the sole cause of diabetic coma; although Roque Dévic found none in the very careful examination he made of his case. The determination of the presence of this acid can only be performed with certainty by a difficult and prolonged chemical process (Wolpe, Külz); but von Noorden suggests that its presence may be inferred with reasonable probability when the urine is lævo-rotatory after trituration with Fehling, fermentation, or precipitation with basic acetate of lead and ammonia.

Duration.—As a general rule diabetes in children and young persons is an acute and rapidly fatal disease, lasting only weeks or months, or at most one or two years; but even in children cases have been known to extend over five, six, or more years. On the other hand, in elderly people the disease usually makes but slow progress, and lasts many years.

Termination.—Death occurs in many cases as a result of one or other of the complications to be described, of which pulmonary phthisis is perhaps the most common. Very often the patient's strength becomes gradually diminished, and he dies quietly in a drowsy condition without actually becoming comatose. But in many cases the accidents that precede death come on more or less suddenly, from some slight cause, such as fatigue, excitement, or a chill; and death is preceded by coma of a peculiar type. Any acute infectious process is peculiarly liable to terminate fatally in diabetes; this has been abundantly illustrated in the last few years owing to the great prevalence of influenza, which has proved fatal to many diabetics, even where no special visceral complications, such as pneumonia, have been manifested; as a rule these cases have died comatose.

Prognosis.—Diabetes is in all cases a grave disease, and the subjects are regarded by all insurance companies as uninsurable lives: life seems to hang by a thread, a thread often cut by a very trifling accident. Still there is something to be said on the subject of prognosis besides the broad rules of age already laid down. In each case we must consider the general condition of the patient, the degree to which treatment is successful in reducing the sugar and maintaining nutrition, and the presence or absence of complications. The state of the circulation, as indicated by the pulse pressure and cardiac impulse, is of great importance. Cases which supervene after injury or acute diseases sometimes get rapidly well, or recover more gradually, so that under these circumstances we may be hopeful at any rate for some time. Other points in favour of the patient are—(i.) the concurrence of obesity or gout; (ii.) favourable social conditions and freedom from business or financial worries; (iii.) early treatment.

Cases have ended in recovery in which the disease appeared to

have been induced by eating excessive quantities of sugar. Such cases should probably be regarded only as temporary glycosuria; nevertheless the glycosuria often tends to recur in such persons, and in them there is probably some underlying tendency to diabetes. Schmidt considers that spontaneous sweating is favourable; but I have observed several instances which did not bear out his opinion. The cases which occur about the climacteric period in women end in recovery perhaps more frequently than other forms of diabetes in elderly people.

A good deal has been said of the prognostic value of the knee-jerks; some observers regard their absence as very grave, but extended experience has disproved this opinion, and the significance of the symptom may easily be overrated (Grube).

On the other hand, where there is a marked family tendency to diabetes or to nervous disease, the ultimate prognosis must be unfavourable. The prospects of patients who are unable to diet themselves properly, to protect themselves from the vicissitudes of the weather, from the hardships of daily toil, or from the cares and anxieties of life, are undoubtedly less favourable; herein, as in most other chronic diseases, a competent income and a tranquil existence count for much in the prognosis.

Complications.—*The Skin.*—Diabetics suffer from many derangements of the skin besides that dryness and roughness of the epidermis which are almost always present. The circulation is feeble; the ears, nose, and cheeks are often cyanosed, and the legs and feet are cold. Elderly patients are peculiarly liable to acne pustules, boils and carbuncles; and a papular erythema on the extremities is one of the common ailments of younger subjects. This last presents rose-coloured, elevated spots, as large as a split pea, thickly distributed over the elbows, knees, wrists, ankles, fingers, and palms of the hands; these often coalesce to form red shiny patches.

Symmetrical erythema may attack the face. Davies Pryce has described an erythematous œdema, which depends, as he believes, on the presence of neuritis. The palms of the hands and the soles of the feet may burn intensely or sweat profusely. The occasional occurrence of general sweating has been already noticed.

Eczema of the genitals, especially in women, may be a most distressing symptom. The disease is undoubtedly set up by the irritation produced by torulæ and other organisms which grow in the saccharine moisture remaining on the parts; but it often results in an intense dermatitis which spreads over the abdomen and thighs, and itches intolerably. It may be prevented by scrupulous cleanliness. Pruritus vulvæ is present sometimes without dermatitis. Purpura may be seen in the early stages.

A horrible affection, described by Kaposi, and called by him papillomatosis diabetica, occurred in the person of a Brazilian patient, and was probably caused by some exotic parasitic growth; the hands and forearms were covered with ulcerating excrescences and warty growths.

Xanthoma diabeticorum, originally described by Addison and Gull,

consists of indurated, rounded, or conical tubercles of a dull reddish colour, their apices being often yellow. The yellow colour is due to the presence of fat globules.

Marchal (de Calvi) relates a case in which the body of the patient was covered with great coppery pustules containing material as hard as very dry cheese. It disappeared, leaving only minute scars, and did not recur. This appears to have been a case of xanthoma, which tends to disappear in the same way.

Little centres of necrosis in the skin about the ankles and dorsum of the feet, first appearing as small round red spots the size of pin-heads, have been described. The disease seems to begin in the sweat-glands.

Cellulitis and gangrene are more apt to occur in diabetes as life advances, and are more frequent in men than in women. Gangrene depends sometimes on vascular disease, necessitating amputation high up; in other cases it is due to neuritis, and may be let alone, or the necrosed parts only removed by amputation.

The condition called by Kaposi gangrena bullosa serpiginosa was probably due to neuritis. The patient, a woman aged 61, had three gangrenous patches on her left leg, and fifteen or twenty bullæ distributed over the neighbouring skin. Perforating ulcer, generally admitted to be due to neuritis, is not uncommon; it is preceded by a circumscribed anæsthetic patch, and when formed the ulcer is surrounded by an anæsthetic zone.

Œdema of the subcutaneous cellular tissue of the lower extremities sometimes is present, and ascites may occur. In the cases of this kind which have come under my observation the dropsy has been due to heart failure, possibly with latent valvular disease.

The temperature in the axilla may be very low. Fagge recorded it as low as 93·6°; and in one of my own cases it was only 95° in the morning, though in the evening it used to rise to 100°.

Nervous system.—It is not uncommon to meet with listlessness and depression of spirits, weakness of mind, and peevishness of temper; definite mental disturbances, as indicated by melancholia with suicidal tendencies or temporary mania, occur more rarely. In some cases the mental disturbance and glycosuria have appeared to alternate. There may be symptoms resembling those due to an intracranial growth; for example, headache and giddiness, epileptic or apoplectic attacks, paralysis of the sixth pair with conjugate deviation of the eyes, ptosis, strabismus or paralysis of a limb, or complete hemiplegia.

Neuralgia is usually symmetrical; it may come on suddenly, after the patient is in bed, the pain being excruciating. Each attack lasts a few hours only, but it may recur at short intervals. Sciatica is very common, and bilateral sciatica is especially suggestive of diabetes.

Some patients present symptoms resembling locomotor ataxy, with ataxic gait, loss of knee-jerks, gastric crises, and lightning pains in the legs. These symptoms are often associated with sciatica, and depend upon a neuritis affecting the sciatic nerves and their branches. The pupillary reflex

was thought to be never abolished, but Grube has shown that this is unfortunately not invariably true, so that the differentiation from true tabes becomes very difficult. The soles of the feet may be intensely hyperæsthetic, and the other phenomena of neuritis may occur, such as hyperidrosis, glossy skin, œdema, ecchymosis, perforating ulcer, loss of nails, and so forth.

Eye affections.—Diabetes causes impaired vision most commonly by weakening the power of the muscles of accommodation; and in the next place by diminishing the perception of light in the retina—diabetic amblyopia. After these changes, in order of frequency, comes cataract, which is usually but not always of the soft variety, and may disappear spontaneously. The retina is liable to be the seat of a peculiar inflammation which takes two principal forms; the cases may be classified as—(i.) Retinitis centralis punctata diabetica; (ii.) Retinitis hæmorrhagica diabetica; (iii.) Mixed forms. In the first the ophthalmoscope shows white glistening patches, not necessarily arranged round the macula, and never fan-shaped, but otherwise bearing a considerable general resemblance to albuminuric retinitis, with which, however, it must not be confounded: in the second the lesion consists of rounded or punctiform hæmorrhages with secondary retinitis; and in the third we have a combination of both changes. Central scotoma, or loss of vision in the central part of the field, is common; and exactly resembles the condition met with in tobacco-smokers. It is probable that tobacco may produce its toxic effect upon the retina more readily in diabetes, but undoubted cases observed in non-smokers have been recorded. The optic nerve is sometimes atrophied, and capillary aneurysms on the retinal vessels have been described. Diabetics also suffer, as I have said, from paralysis of the ocular muscles; and hemiopia, various inflammatory affections of the ocular structures, conjunctivitis, keratitis, iritis, and choroiditis are met with.

Other special sense affections.—Blunting of the senses of smell and taste has been recorded in one case. Deafness, due to otitis media, or to œdematous swelling of the Eustachian lining, may occur. Diabetic otitis media is a very acute inflammation, coming on suddenly without any previous coryza or cold; it is characterised by severe external pain in the mastoid region, tinnitus and extreme deafness. The auditory canal becomes red and swollen, secreting muco-pus; the tympanum is congested, œdematous, and dull; the mastoid cells are infiltrated with pus, and the osseous tissue may be extensively destroyed. According to Raynaud, the disease begins in the bone. Fever may be entirely absent. In some cases true nerve deafness has been observed.

Respiratory system.—A case of acute and rapidly fatal membranous inflammation of the larynx and trachea attacking a male patient under treatment for diabetes in the Royal Infirmary of Edinburgh was reported by the late Dr. Warburton Begbie; but with this exception no mention of any complications affecting these structures has come under my notice, and it is possible that this was merely an example of diphtheria occurring in the course of diabetes. The bronchial tubes may be affected by catarrh,

and this may precede the appearance of pulmonary phthisis, which is one of the most common complications. Since the discovery of the tubercle bacillus has placed within our reach an easy means of determining whether a pulmonary lesion be tubercular or not, it has become clear that non-tubercular cases are much more rare than was formerly supposed; there can be no doubt, however, that they do sometimes occur. I have had three cases in which I could find no tubercle bacilli, but a more conclusive case with careful post-mortem examination has been recorded by Roque Dévic and Huguenenq. The disease comes on as a rule insidiously, and is often attended by little cough or rise of temperature. It may, however, follow an attack of acute lobar pneumonia. Such attacks of pneumonia in diabetic patients often prove fatal, but by no means invariably so; complete recovery may take place, or permanent lung mischief may follow. Gangrene may supervene upon acute or chronic inflammation of the lungs, and is more often met with in diabetes than in any other constitutional state. It is often undetected during life, as the characteristic offensive odour of the sputa may be entirely absent.

Circulatory system.—Diabetic endocarditis is a rare complication; it most commonly affects the mitral valves, rarely those of the aortic opening. Heart failure, on the other hand, due to fatty and fibroid degeneration, is a common occurrence, and may be regarded almost as part of the ordinary course of the disease. It may be associated with attacks of dyspnœa or faintness, or of angina pectoris; and such symptoms should be regarded as indications of the danger of sudden death. It is therefore of great importance to watch the state of the circulation, and to observe the character of the pulse. Elderly patients are not exempt from the ordinary senile changes in the vessels, and these when seated in the extremities may lead to gangrene.

Digestive system.—Loosening and loss of the teeth, from atrophy of the gums, gingivitis, and spongy or bleeding gums, are common; the teeth themselves are also very apt to be attacked by caries, causing toothache, abscess of the root, and the like. Loss of sleep from toothache is a very serious matter to a diabetic, and careful attention to the hygiene of the mouth is most important. The saliva is generally diminished, so that the mouth is dry, and sour eructations are not uncommon. Hunger is one of the symptoms of diabetes, and sometimes a feeling of emptiness at the pit of the stomach is troublesome. As a rule diabetic patients do not suffer from painful digestion, but the tongue is often furred, and post-mortem examination shows the frequency of gastritis. Some patients are liable to periodical attacks of gastritis, which may be attended by jaundice or diarrhœa; and according to Grube gastric crises, resembling those of tabes, are by no means rare. Examination of the stomach functions does not indicate that there is any greater disturbance of the digestive capacity than might be expected to result from the chronic gastritis. Hirschfeld has described cases which were characterised by early attacks of pains, with light-coloured, fatty stools, suggestive of pancreatic colic; and these may be compared with Fleiner's

cases, already quoted, in which diabetes supervened years after the attacks of pancreatic colic. Diarrhœa is sometimes very troublesome, needing considerable care in diet. Unfortunately the diabetic diet is not very digestible; the bran, almond, or cocoa-nut biscuits, green vegetables and salads, prescribed for these patients as substitutes for bread and potatoes, contain a large amount of indigestible vegetable material which acts as a mechanical irritant to the gastro-intestinal mucous membrane.

Enteric Fever.—Diabetic patients appear to be more than ordinarily susceptible to the poison of enteric fever, but the attacks are usually mild in type. During the fever the sugar generally disappears. Dr. Noel Paton has shown that increased elimination of heat in animals favours glycogenesis, but that the products of the growth of micro-organisms inhibit it; to the latter, then, we must ascribe the disappearance of the sugar.

Rheumatism.—Rheumatic muscular pains are very common in diabetes, and thickening and shortening of the palmar aponeurosis (Dupuytren's contraction) has been repeatedly noticed; though it is probably due to a rheumatic or gouty condition of the patient and not to diabetes.

New Growths.—Some authorities believe that diabetes disposes to the formation of tumours, but that when they occur they grow slowly; the grounds for these opinions are not very clear.

Diabetic Coma.—The liability of diabetes to terminate suddenly in a peculiar form of coma has been known for many years, but its special features were first described in 1874 by Küssmaul, whose name has been attached to the affection.

Attention to the phenomena in question was first aroused in England by Sir Walter Foster's paper at the Manchester meeting of the British Medical Association in 1877.

Küssmaul's coma occurs at all ages, and in diabetics of both sexes, but with much greater frequency in the young. The direct causes are fatigue, excitement, exposure to cold, and any intercurrent acute disease. The remoter causes are absence of starchy food and constipation. Its pathology is still obscure; Küssmaul's original suggestion that it is due to poisoning by acetone has not been proved, and careful analysis has failed to detect the presence of this substance in the blood of some persons dying in this manner.

The symptoms resemble those produced in animals by large doses of dilute acids, and considerable quantities of acid have been shown to be present in the blood. This acid has been identified by Minkowski as β-oxybutyric acid, a substance which breaks up to form ethyl-diacetic acid which, on further decomposition, yields acetone and carbonic anhydride. As I have already said, Professor von Noorden regards β-oxybutyric acid as the true cause of the coma; but this can hardly be admitted in the face of the failure of Roque Dévic and Huguenenq to find any in the exhaustive examination of their case.

Dr. Latham has suggested that the poison may be paraldehyde, and

has even proposed a theory of its formation; Senator has suggested trimethylamine, but unfortunately neither poison has been demonstrated.

A few eminent authorities still regard the phenomena as uræmic; but, as the description will show, the classical symptoms of uræmic coma are different in many respects. In diabetes the onset of coma may be preceded by malaise, languor, and weakness of some days' duration; there may be a gradual fall in the quantity and specific gravity of the urine, and this may give the reaction for acetone and with ferric chloride. Accelerated or disturbed respiration is a warning symptom which may be noted in advance by the physician. But in many instances the attack is ushered in without previous warning by restlessness, delirium, or even maniacal excitement; or the patient may complain of weakness only. The pulse becomes very rapid and feeble, the epigastrium painful, and the respiration more hurried and deeper. In course of time drowsiness sets in, which deepens into coma; the patient then lies quietly in bed, breathing 30 or 40 per minute, with a deep sighing respiration; the pulse beats 130 to 150 per minute, and is very feeble; the face is usually pale, the body and extremities cold, and the temperature subnormal. The secretion of urine is diminished or suppressed, and the bowels can be got to act with difficulty. Death is sometimes preceded by a rise in temperature (103°-104° F.), by convulsions, or by cyanosis. A peculiar odour of the breath is often present, but is not constant; it varies considerably in character, for it has been diversely likened to sour beer, rotten apples, hay, chloroform, and so on. The urine always contains acetone, and is generally but not constantly albuminous; the ferric chloride reaction is, as a rule, present, but exceptions to this rule have been observed.

The symptoms, as is indicated by the above description, though fairly uniform, do not always present the same picture; the late Professor Frerichs endeavoured, not altogether successfully, to distinguish three groups, but it is doubtful whether there is either real utility or pathological justification for such a classification. It is of more interest to note that this form of coma is not peculiar to diabetes, but has been observed in pyonephrosis, chronic cystitis, gastric and hepatic cancer, anæmia, etc.

Acetonuria is not of itself a certain indication of the imminence of this grave complication, as it has been often met with in patients who have lived for many months without such an occurrence. Hirschfeld suggests that in many cases this symptom is caused by the want of non-nitrogenous food, and that where this is the case it may be removed by the addition of a certain proportion of carbohydrates to the diet. He thinks this precaution should always be taken when acetonuria occurs, so that the occasional may be distinguished from the persistent cases, and also because the continuance of strict diet may in itself be a danger. His statements have been verified to a slight extent, but not sufficiently to enable us to assert that we have in this test a sure or even a useful means of prognosis. As will be seen when we come to speak of the treatment of diabetes, there is at the present time a salutary reaction against the too stringent

diet of former years, so that there is now less probability of this kind of auto-intoxication arising from defective diet alone; and Küssmaul's coma has often come on in cases of diabetes in which, either by the care of the physician or by the wilfulness or neglect of the patient, a mixed diet had been taken. It is a very grave symptom, and although in view of the recoveries claimed by Reynolds, Quincke, Gamgee, and others, it may not be invariably fatal, recovery is extremely rare. Less uncommonly we see the premonitory symptoms, such as epigastric pain, sighing, breathing, and even some degree of drowsiness, pass away; in one case under my care this disappearance followed an attack of spontaneous diarrhœa, and, besides this, another instance of recovery from epigastric pains and drowsiness is recorded in my lectures.

Treatment.—Until the pathology of diabetes has been made perfectly clear, the best guides to treatment must be found in those generalisations from experience which have accumulated since the diagnosis has rested on a sure foundation. We have acquired much useful knowledge concerning the management of diet, the use of drugs, general hygiene, and so forth; but above all, it is important to bear in mind that adherence to blind routine may be most disastrous, and that we are bound to study the needs of each individual case. Some patients are benefited by means which are harmful to others, and in many instances life may be shortened by the vigorous application of rules which may be salutary for the majority. This may appear to be a self-evident proposition, but if so, there is none the less need to call attention to it, as the ordinary practice seems to be to copy a dietary out of a text-book, and hand it to the patient with a prescription of some preparation of opium, or of one of its alkaloids.

Diet.—In previous sections it has been shown to be most probable that the excess of sugar in the blood, upon which nearly all the symptoms of diabetes depend, is due to its over-production in the liver; and although it is tolerably certain that sugar may be derived from nitrogenous sources, we must admit that carbohydrate food constitutes its chief source of supply, and the one most readily controlled. It has therefore been established as the cardinal principle of the treatment of diabetes to restrict those articles of diet which have this chemical composition. But experience has shown that this rule must not be applied too rigidly; under it some patients lose weight and become weak; and acetone appears in their urine. The importance of giving diabetic patients as much carbohydrate as they can assimilate has been recognised by several modern writers, especially by Grube. Hirschfeld has shown that in moderate cases the addition of a certain amount of carbohydrate to the diet causes acetonuria to diminish or disappear; and Leo found that it preserves the albumin of the body, though to a less extent than in health.

We have passed through various phases in the dietetic treatment of diabetes. Rollo began by feeding his patients exclusively on meat; this proved harmful and impracticable, yet until recently it has been the rule to allow the patient as little carbohydrate as he would submit

to be deprived of, a practice that pressed very hardly on the more docile and conscientious, but was evaded by the unscrupulous or careless. My practice permits each patient to enjoy the maximum amount of carbohydrates that he can assimilate. In order to ascertain this I first determine the extent to which the sugar excretion depends on carbohydrate food, and for this purpose I put the patient for a week on strict diet such as this :—

Meat soup; beef tea. Fish of all kinds. Butcher's meat. Poultry; game; eggs. Green vegetables; sea-kale; celery; salads. Milk; cream; custard. Butter; cheese; cream cheese. Jelly; isinglass; Irish moss. Tea; coffee; Hollands; whisky; mineral waters.

These articles must be free from sugar, and of course no sugar must be used in preparing any of them for the table; the milk must be limited to a pint or a pint and a half daily. In addition we may allow, as a bread substitute, almond bread or biscuits, such as are now made of well-ground almond meal, free from starch; and sponge-cakes prepared from the same meal may be taken with tea. Saccharine (glusidum) should be used for sweetening any of these articles. Five days are required to free the liver of a starved animal from glycogen, so that after a week of this diet we may expect to obtain urine fairly representative of its sugar-forming capacity. At the end of that time the urine should be collected for twenty-four hours, mixed, measured, and the total output of sugar estimated in grains by quantitative analysis. The result varies with the gravity of the case. In mild cases there may be no sugar, or one or two hundred grains only; in more serious cases the reduction is only to half or one-third of the previous amount. But the good effect of the change in the diet shows itself in a great improvement in most of the symptoms: the frequent micturition ceases to trouble the patient, so that he gets almost unbroken sleep; his thirst disappears, and the quantity of urine falls nearly to normal limits. In consequence of this relief his general condition becomes decidedly better, and he regains part of his lost vigour. This result has, however, been obtained by a certain amount of sacrifice on the part of the patient, and it is most desirable to endeavour to make his condition as tolerable as possible by any concessions which will not do him harm.

When we are satisfied that we have obtained the figure indicative of the maximum effect possible by rigid diet, we may allow, in addition to the above enumerated articles, one large baked potato daily. (It is important that it should be baked, and not boiled.) At the end of another week the urine is collected as before, and the sugar again estimated. The result is almost invariably to show that no additional sugar excretion has taken place. We may then proceed by permitting two potatoes daily, watching the urine at the end of a week; but our patients often complain most of the deprivation of bread. Since the introduction of gluten bread sixty years ago by Bouchardat it has been the principal means used to satisfy this craving. But the best gluten bread contains at least 25 per cent of starch, and many samples contain 40 per cent;

indeed it is impossible to bake it with less than the minimum stated, so that, as ordinary wheat bread contains about 42 per cent of starch, it is plain that so far as the starch is concerned our patients might just as well eat half the weight of ordinary bread. This unpalatable and expensive substitute cannot therefore be regarded as satisfactory. For some years past it has been declining in favour; and it is better to give ordinary bread in the form of very dry toast in definite quantities, namely, 1½ oz. with each meal, or 4½ oz. daily. Bread must not, however, be allowed in grave cases of diabetes, as even a small quantity appears to bring back all the acute symptoms; for such patients we must be content to order bread, cakes, and biscuits made of almond flour, or cocoa-nut flour, which may be lightened by a very little gluten flour.

Unfortunately many articles, prepared and unprepared, but purporting to be free from starch and sugar are not what they are represented to be; and every practitioner who undertakes the charge of a diabetic should make himself familiar with the simple means by which to detect the presence of these admixtures. In many instances this may be done by merely dropping a little weak iodine solution on the biscuit, bread, or flour. In a recent instance biscuits were sold as containing 2 per cent of starch, but which, on the addition of iodine, turned nearly black and proved on analysis to contain 46 per cent of this carbohydrate. The so-called gluten bread often contains nearly as much. But this test does not detect sugar, and the simplest means to detect the presence of carbohydrates is to boil the substance with dilute sulphuric acid, neutralise with caustic potash, and test with Fehling's solution. If all practitioners would protect their patients by doing this, or getting this done before sanctioning any diabetic bread substitute, these fraudulent articles would be speedily driven out of the market. It is to be regretted that "Soy" flour should be recommended by some writers as free from carbohydrates, for it really contains about 24 per cent (Kinch),[1] while some Soy biscuits sold for the use of diabetics contain twice as much. Even almond flour and desiccated cocoa-nut may contain sugar, are sometimes adulterated by the addition of starch, and without analysis are not to be regarded as safe. The following directions for making cakes may be useful (85*a*):—

Almond cakes: take ground almonds, 1 lb.; 4 eggs; two tablespoonfuls of milk; a pinch of salt (or saccharine): beat up the eggs, and stir in the almond flour; divide into cakes, and bake in a moderate oven for 45 minutes.

Cocoa-nut cakes: take finest desiccated cocoa-nut, ¾ lb.; ground almonds, ¾ lb.; 6 eggs; ½ teacupful of milk: mix and divide, and bake for 25 minutes. The addition of gluten flour makes lighter cakes, but involves the presence of some starch; it is also an improvement to ferment the dough.

[1] The following analysis of Soy beans by Prof. Kinch is to be found in Frankland's *Agricultural Chemistry*, p. 198:—Water, 11·3; nitrogenous matter, 37·8; fat, 20·9; carbohydrates, 24·0; fibre, 2·2; ash, 3·8.

In accordance with the principle of permitting all carbohydrates to be taken that do no harm, the observations of Külz, made as long ago as 1874, that diabetics could assimilate lævulose and inuline, have borne fruit in the production of commercial lævulose for diabetics. Two preparations are in the market—one a granular powder, made by Schering and Glätz of Berlin, the other a treacle-like substance, sold by Allen and Hanbury. Either of these may be ordered for diabetics in quantities up to 1½ oz. daily, and are usually assimilated without causing any increased glycosuria. It is noteworthy that lævulose is not excreted as such, but appears to be converted into dextrose (glucose). Inuline, a form of starch found in dahlia tubers, cannot be obtained commercially except as the tubers; but Dr. Hale White suggests that these may be cooked and eaten as a vegetable.

The use of alcoholic drinks is not necessary for any one, and certainly not for persons suffering from diabetes; it is very doubtful whether, as suggested by some authors, alcohol supplies the deficiency in carbohydrates: many patients, however, require some concession to be made to their previous habits. Scotch and Irish whisky and Hollands gin are, as a rule, free from sugar. Still wines from the Rhine and Moselle districts, and light Bordeaux wines (vin ordinaire), contain very little; Burton bitter ale is nearly deprived of all sugar. A little whisky or Hollands, or one or two glasses of the above-mentioned light wines diluted with alkaline mineral water, may be allowed. Sugar-free champagne is advertised for sale. With respect to beer it must be remembered that it is usual to drink it by the pint, so that its use can only be sanctioned in particular cases, and its effect on the urine should be watched with care.

Climate and health resorts.—There are, perhaps, many climates more enjoyable for an invalid than those of the British Islands, and there are several famous health resorts which offer special facilities for the treatment of diabetes; but diabetic patients do not bear well the fatigues and excitement of travelling, and it is the universal experience of physicians that too often they leave home to find a grave in a foreign land. No patient suffering acutely should be sent away from home, even for a comparatively short distance, if it can possibly be avoided; on the other hand, there is a large class of chronic patients who derive great benefit from change of climate or a course of treatment at one of the foreign spas. Speaking generally, a diabetic patient will do best in an equable, sunny, moderately dry, and not too hot climate; but as it is hardly possible to find these conditions in the same place all the year round, a change is needed according to the season. Moreover diabetics, like other people, have their idiosyncrasies, and these must not be lost sight of. Those who are braced up and strengthened by mountain air should not be sent to the sea, and conversely.

The mineral water stations which have acquired a great reputation in the treatment of diabetes are Vichy, Contrexéville, Carlsbad, and Neuenahr.

The waters of Vichy are all of much the same composition, and contain bicarbonate of soda as their principal constituent. Their use is undoubtedly beneficial in diabetes, in which disease the alkalinity of the blood is diminished; there is some reason, indeed, as we have just seen, to believe that this diminution is the chief cause of diabetic coma [*vide* art. "Balneo-Therapeutics," vol. i. p. 335]. A bottle of Vichy water daily, taken with a little light wine or lemon juice, is a prescription which is often useful, and one which can be followed in any part of the world. Vichy is a summer station, the season being from April to September; but as it is too hot in July and August, the best months to select for a visit are May and June.

Contrexéville is situated in the Vosges, and is very suitable for gouty diabetics in the autumn months; the water chiefly contains bicarbonate of calcium; its taste is pleasant, and it makes a very good table water. Carlsbad, in spite of its great vogue, probably owes its reputation less to the specific action of its waters than to the skill of the many eminent physicians who practise there during the season. Carlsbad water contains mainly sulphate of sodium, and is actively aperient. The journey thither is very long and fatiguing, even when accomplished by easy stages, and with all the comforts that money can ensure; the town, when reached, is beautifully situated, and in the early summer its surroundings offer many attractions to the visitor. The waters are decidedly useful if it be desired to overcome constipation or to treat obstinate stomach catarrh, or if diabetes be associated with marked obesity. The season lasts from April 15th to September 15th.

Neuenahr (Ahr valley, Rhine) is much more readily accessible from England than Carlsbad. Its waters contain sodium chloride, sodium carbonate, and carbonate of iron. The treatment of diabetes is carried out with much success at this quiet, pretty, semi-rural watering-place. The season, like that of Carlsbad, is from April 15th to September 15th; but July is better avoided. There are many waters which may be used by diabetics at home. Those of Vals, La Bourboule, Royat, Giesshübel, Apollinaris, Johannisquelle, and Bethesda may be employed, like those of Vichy or Contrexéville, as table waters. On the other hand, the waters of Marienbad, Chatel-Guyon, and Rubinat are aperient, and resemble Carlsbad water in that they depend for this property on the large proportion of sodium sulphate which they contain. Rubinat is especially useful to overcome the obstinate constipation which is so frequent and troublesome a complication of diabetes.

For the winter months, if it is desirable to send the patient away from home, the various watering-places on our own south coast, or in the Riviera, or Montreux, Arcachon, Biarritz, Algiers, Egypt, or the Canary Islands, offer in varying degrees the advantages of a warmer climate and change of scene; but in the majority of cases it is not necessary to send the patient away from home for the winter; as a rule diabetics do better when surrounded by the comforts of their own firesides.

Exercise and Massage.—I wish to lay especial stress upon the opinion

that a diabetic should live as much as possible in the open air; and for this purpose walking, cycling, riding, driving, and such out-of-door games as golf should as far as possible be encouraged. At the same time we must warn our patients against the dangers of over-fatigue and catching cold. When indoors the rooms occupied must be well ventilated and plentifully supplied with pure air. The body must be suitably clothed, preferably in light woollen garments; and in this respect there has been of late years a great advance towards a proper perception of the rational in dress. Late hours, hot crowded gatherings, excitement, and, in severe cases, even games in which the danger of fatigue or over-exertion is lost sight of in the desire to win, must be strictly forbidden. When from any temporary cause the bodily weakness is too great to permit of active exercise, massage may be usefully employed to promote nutrition and restore vigour to the muscles.

Baths and Bathing.—Bathing of all kinds may be allowed if it is not too prolonged, and does not involve violent exertion, as in swimming in a rough sea. The daily sponge bath is not contra-indicated, and its temperature must depend upon the state of the patient. Warm baths and steam baths are much appreciated by those who have dry and irritable skins. The Turkish bath is useful in obesity when to some extent it fulfils the purpose of exercise.

Drugs.—Opium and its alkaloids possess a well-deserved reputation in the treatment of diabetes, and hold the front rank. They appear to have greater power than any other known remedy over the sugar-forming function of the liver; but this power is limited, and its extent is readily ascertained in each case. While it is easy to push the dose up to almost any amount, the results obtained do not support this practice, which is attended by the usual ill effects of the opium habit. Where we desire to diminish the total amount of urine secreted as rapidly as possible, a grain of extract of opium in pill may be ordered at bed-time; and it is permissible to give even more frequent doses of this remedy, or of morphine, for a time. But this should be done with discrimination, and the practitioner must not rely too much upon its influence.

Codeia is a weak substitute for opium or morphine, and has little utility except as a placebo. Belladonna is sometimes combined with opium, and the combination acts well.

Salicylate of sodium has been credited with several cures (Sympson), but it is doubtful if it possesses any specific action; the dose is from thirty to sixty grains daily.

Bromide of potassium exerts a favourable influence in soothing the nervous system, and is sometimes a valuable agent; the dose usually given is thirty grains daily.

Jambul, in the form of powder, extract, or tincture of the seeds of Syzygium jambolanum, is a drug which has great repute in the East Indies and Malayan Archipelago, but it has not justified this reputation in Europe.

Arsenic is a valuable tonic, and arsenite of bromine, or rather liquor

Clementis,[1] has been counted as a specific. Its value is at best doubtful.

The success of thyroid extract in the treatment of myxœdema gave a great impetus to therapeutic experiments with organic extracts in other diseases; and following the indication afforded by the recent observations on the influence of extirpation of the pancreas, numerous attempts to treat diabetes have been made by administering raw pancreas, or various preparations derived from the gland, to diabetics. The result has been very disappointing. No good whatever has been effected by these means (H. W. G. Mackenzie (68)). Even depancreatised dogs have not been benefited by it (Minkowski, Gley, and Thiroloix). Lépine has recommended hypodermic injections of pilocarpine to stimulate the pancreatic secretion, but with little success, if any. He has produced, however, from malt diastase, a glycolytic ferment which has been administered to several patients with good effect, the elimination of sugar being reduced in each case. He makes the ferment by macerating five grammes of malt diastase, obtained as pure as possible, in a litre of water acidulated with one gramme of sulphuric acid. After being allowed to digest for two or three hours it is neutralised with bicarbonate of soda, and this quantity (a litre) is administered daily. As it will not keep it must be made fresh every day.

Dr. Karl Grube of Neuenahr has recently referred to the popular use of egg-shell powder in diabetes, and has seen general improvement without influence on the glycosuria follow the use of a teaspoonful daily of this remedy, or an artificial substitute consisting of carbonate of lime 93 parts, phosphate of lime and phosphate of magnesia 7 parts. He refers to the great loss of lime in the form of earthy phosphate in the urine, and thinks the medication may tend to repair that waste.

Among other remedies recommended are Martineau's specific;[2] antipyrin in 15-grain doses thrice daily (Germain Sée); sulphide of calcium, $\frac{1}{4}$ to $\frac{1}{2}$ grain three or four times in twenty-four hours (Cauldwell); creasote, 4 to 10 drops daily (Valentine); phenacetin and exalgin (Dujardin-Beaumetz); camphor (Peyrand); iodoform (Moleschott); nitro-glycerine (Kennedy); salicin, 30 to 270 grains daily (Dornblüth); carbonate of soda, $\frac{1}{2}$ oz. to 2 oz. daily, with citric acid (Stadelmann); sulpho-carbolate of soda, 5 to 30 grains for a dose (Monckton); nitric or nitro-hydrochloric acid with tincture of nux vomica (Wilks); cocaine, $\frac{1}{4}$ grain three times a day (T. Oliver); pepsin, 10 grains three times daily (Gardner); ouabaïne, $\frac{1}{1000}$ gr. three or four times daily (Gemmel); phos-

[1] Liq. Clementis, according to Hager, has the following composition: Acidi arseniosi, Potassii carbonatis āā gr. iss. Dissolve in five drops of distilled water in a test-tube, and warm until a clear solution is formed. Dilute with distilled water until the quantity weighs 153 grains. Then add 6·2 grains (4 drops) of bromine, and set aside for one day. The fluid is then ready for dispensing.

[2] Martineau's specific:—Carbonate of lithium, 20 centigrammes (3 grains), added to a tablespoonful of the following solution: Arseniate of sodium, 0·20 centigramme ($\frac{1}{30}$ grain); distilled water, 500 centigrammes (80 minims); the mixture to be placed in an ordinary soda-water machine (gasogene), this quantity to be used daily with and between meals as a drink, alone or mixed with wine.

phorus, gr. $\frac{1}{30}$ in pearls, of which three to six are to be taken daily (Balmanno Squire); lactic acid (Cantani); hydrogen peroxide; uranium nitrate; liquid extract of ergot in half-drachm doses thrice daily (Dougall); benzosol, 60 to 75 grains daily (Piatkowski); strontium bromide, 30 grains thrice daily (Solis Cohen); permanganate of potash and maté (Monin); piperazine (Hildebrandt).

Dr. S. West has endeavoured to restore our confidence in uranium nitrate administered in doses up to ten grains three times a day. It was formerly used in doses of only one or two grains, and is liable to cause gastric irritation. A certain experience of the larger doses has assured me that even these have no constant or specific influence on the disease.

Treatment of symptoms and complications.—Neuralgia.—The sciatic and other pains often cease as the disease is controlled by general treatment, but the presence of these symptoms affords a justification for the use of opium or hypodermic injections of morphia. Quinine in 5-grain doses, or phenacetin, or antipyrin in 15-grain doses may be tried. Butyl chloral hydrate in 15-grain doses is particularly effective for facial neuralgia. The part may be painted with a liniment of equal parts of menthol (or camphor) and chloral, or the following ointment may be applied:—℞ Menthol gr. xv., Cocainæ hydrochlor. gr. v., Chloral hydratis gr. iij., Vaselini ʒj.

Thirst.—This, too, is relieved as a rule by means which diminish the polyuria. It is useless to place restrictions on the patient's drink, but sucking pieces of lemon, or ice, or sipping very hot water, may diminish the demand, while hypodermic injections of pilocarpine stimulate the flow of saliva and relieve the dryness of mouth; glycerinum acidi carbolici in 5-minim doses is said to have the same effect. If it is desired to give lactic acid, this may be ordered as a refreshing drink in the form of lemonade, as follows:—℞ Acidi lactici ℥ss., Tr. limonis ℥iss., Saccharini solub. gr. iv., Aquam ad Oj. Constipation in some cases appears to increase the thirst, and is almost always a cause of discomfort, if not of danger, from the absorption of the products of pancreatic digestion and intestinal fermentation. Any laxative which does not contain sugar may be employed, but the best remedies are the aperient mineral waters, especially those containing sulphate of sodium, of which Rubinat is the strongest and most effective. There are also the waters of Condol, Carlsbad, Chatel-Guyon; and of those containing sulphate of magnesium, Franz-Joseph, Hunyadi Janos, Freidrichshall, Æsculap, and so on.

Œdema generally disappears rapidly after rest in bed. In obstinate cases massage and bandaging should be tried. Dr. Dickinson believes that the tincture of perchloride of iron in doses of 30 to 40 minims three or four times a day acts as a specific in this respect.

Pruritus.—This very troublesome affection in women can only be cured by great attention to cleanliness. After micturition the patient should always wipe the parts with a sponge wrung out of a saturated solution of borax; and for this purpose she should carry the sponge, when away from home, in a small sponge bag under her skirts. It

should be frequently washed in hot water, and wrung out of a disinfecting fluid. The parts, if sore, should be bathed with warm saturated solution of borax, then carefully dried, and powdered with boracic acid 1 part, French chalk 3 parts, or ointment of boracic acid, zinc, cocaine, or iodoform may be applied instead of the powder. The greasy applications may be more protective against the drip of the urine. The vulva should be washed twice a day at least with warm soap and water.

Sweating, where excessive, may be checked by the use of Dover's powder as a substitute for other forms of opium in the general treatment, and by the combination of opium and belladonna mentioned above.

Coma.—There is no remedy for this most serious complication. At the first indication of its onset the bowels should be got to act, if possible, by a brisk purge of compound jalap powder; in addition, citrate of potash, as recommended by E. S. Reynolds, in doses of half a drachm to a drachm, dissolved in copious draughts of water, may be given every hour; but neither these means nor the injection of normal salt solution into the veins, under the skin or into the peritoneal cavity, nor inhalation of oxygen, nor hypodermic injection of strychnine, ether, or camphor, has in the long run proved to be of the slightest use.

ROBERT SAUNDBY.

REFERENCES

TEXT-BOOKS.—1. DICKINSON, W. H. *Diabetes.* London, 1877.—2. LECORCHÉ, E. *Traité du Diabète.* Paris, 1877.—3. NOORDEN, C. VON. Art. "Diabetes," *Twentieth Century Practice of Medicine*, vol. ii. London, 1895.—4. PAVY, F. W. *Researches on the Nature and Treatment of Diabetes.* 2nd ed. London, 1869.—5. PURDY, C. W. *Diabetes.* Philadelphia and London, 1890.—6. SAUNDBY, R. *Lectures on Diabetes.* Bristol and London, 1891.—7. SEEGEN, J. *Der Diabetes mellitus.* Berlin, 1893.—8. SENATOR, H. Art. "Diabetes," Ziemssen's *Cyclopædia*, vol. xvi.

9. ADDISON and GULL. "On a certain Affection of the Skin, Vitiligoidea—a. plana, b. tuberosa," *Guy's Hospital Reports*, 1851.—10. ARMANNI and CANTANI. *Le Diabète sucré et son traitement diététique.* Paris, 1876.—11. ARTHUS, M. "Glycolise dans le sang et ferment glycolitique," *Arch. de Phys.* 1891, No. 3.—12. BANATVALA, H. E. "The Use of Syzygium Jambolanum in Diabetes," *Mid. Med. Miscell.* 1882, vol. i. p. 205.—13. BAUMEL, L. *Pancréas et Diabète*, Montpellier Méd.-1881-82; *Étiologie et pathogénie du Diabète sucré*, Communication faite au Congrès Méd. de Lyon le 25 Oct. 1894.—14. BEGBIE, J. W. "A Case of fatal Croup in the Adult," Collected Works. New Syd. Soc. 1882.—15. BERNARD, CL. *Leçons sur le Diabète.* Paris, 1855.—16. BERTILLON, J. *De la fréquence des principales maladies à Paris pendant la période* 1865-87, Paris, 1889; and an unpublished letter to Dr. Saundby.—17. BOUCHARDAT, A. "Nouveau recherche sur le diabète," *Compt. rendus*, 1841.—18. BREMER, L. "Ueber eine Färbemethode, mit der man Diabetes und Glycosurie aus dem Blute diagnostiren kann," *Centrlblt. f. d. med. Wissensch.* Dec. 8th, 1894.—19. BUTEL. "The Relation between Urea, Sugar, and Phosphoric Acid in the Urine," *Journ. des Connaissances méd.* 1887.—20. CAULDWELL, C. M. "Calcium Sulphide in the Treatment of Diabetes mellitus," *New York Med. Journ.* 1884, vol. i. p. 380.—21. CAVAZZANI, A. "Morbid Changes in the Sympathetic System in Diabetes mellitus," *Centrlblt. f. allgem. Path.* July 1st, 1893.—22. CHARRIN and CARNOT. "Diabetes and Microbes," *The Med. Week*, 1894, pp. 259 and 532.—23. CHAUVEAU and KAUFMANN. "Sur la pathogénie du Diabète," *Le Progrès Méd.* 1893, vol. ii. p. 120.—24. CHURTON, T. "A Case of Diabetes associated with Pancreatic Cyst," *The Lancet*, June 2nd, 1894.—25. DEBOVE. "The Contagiousness of Diabetes," *Bull. Soc. Méd. des Hôp., Journ. de Méd.* Sept. 29th, 1889.—26. DOBSON, M. "Experiments and Observations on the Urine in Diabetes," *Med. Obs. and Enquiries*, vol. v. London, 1776.—27. DOUGALL, J.

"Ergot in Diabetes mellitus," *Glasgow Medical Journal*, vol. xxxix. p. 96.—28. DUJARDIN-BEAUMETZ. "Du régime alimentaire dans le Diabète," *Bull. de Thérap.* vol. iii. p. 385.—29. DUNCAN, A. *Clinical Reports*, Case 28, p. 137. London, 1818.—30. EAGER, T. C. "A Case of Diabetes treated by Pilocarpine," *The Lancet*, 1884, vol. ii. p. 275.—31. EBSTEIN, W. *Ueber die Lebensweise der Zuckerkranken.* Wiesbaden, 1892.—32. FAGGE, C. H. *The Principles and Practice of Medicine*, 1886.—33. FLEINER, W. "Zur Pathologie des calculösen und der arteriosclerotischen Pankreas-cirrhose und der entsprechenden Diabetes-formen," *Berlin. klin. Wochenschr.* 1894, Nos. 1 and 2.—34. FOSTER, B. W. "Diabetic Coma—Acetonæmia," *Brit. Med. Jour.* 1878, vol. i. p. 78.—35. FRERICHS, T. *Ueber den Diabetes*, Berlin, 1884; "Plötzlicher Tod und Coma bei Diabetes," *Zeitschr. f. k. Med.* Bd. vi. Heft 2, 1883.—36. GAGLIO. *Riforma medica*, 1891, No. 2.—37. GANS, E. "Ueber das Verhalten der Magenfunctionen bei Diabetes mellitus," *Verhandl. d. Congr. f. inn. Med.* 1890.—38. GARDNER, E. B. "Pepsin in Diabetes," *The Practitioner*, 1886, vol. xxxvii. p. 448.—39. GEMMELL, W. "On Ouabain in Whooping-cough," *Brit. Med. Journ.* 1890, vol. i. p. 950.—40. GRUBE, K. "Ueber das Verhalten des Patellar-reflexes bei Diabetes mellitus," *Neurolog. Centrlblt.* 1893, No. 22; "Tabes oder Diabetes," *Neurolog. Centrlblt.* Jan. 1, 1895; "On the Treatment of Diabetes mellitus, especially of its more advanced kinds," *The Lancet*, vol. ii. p. 1613; "Beobachtungen über die Bedeutung des Kalkes bei Diabetes mellitus," *Munch. med. Wochenschr.* 1895, No. 22; "Gastrische Krisen bei Diabetes mellitus, nebst Bemerkungen über die Behandlung der Verdauungsstörungen der Diabetiker," *Munchen. med. Wochenschr.* vol. xlii. 1895, p. 136.—41. HUDELMANN. "Ueber die Ursachen der pathologischen Ammoniakausscheidung beim Diabetes mellitus und des Coma diabeticum," *Arch. f. exp. Path.* xvii. p. 419.—42. HAGER. "Composition of Liq. Clementis," *Wiener med. Blätter*, 1883.—43. HALLERVORDEN, E. "Ueber Ausscheidung von Ammoniak im Urin bei pathologischen Zuständen," *Arch. f. exp. Path. u. Pharm.* xii. 1880, p. 237.—44. HANOT and SCHACHMANN. "Sur la Cirrhose pigmentaire dans le Diabète sucré," *Arch. de Physiol.* 1886, vol. i. p. 50.—45. HANSEMANN. "The Pancreas and Diabetes." Int. Med. Congress. *Brit. Med. Journ.* 1894, vol. i. p. 796.—46. HARLEY, VAUGHAN. "The Pathogenesis of Pancreatic Diabetes mellitus," *Brit. Med. Journ.* 1892, vol. ii. p. 452.—47. HARRISON, E., and SLATER, C. "On the Relations of the Amounts of Sugar and Urea in some Cases of Diabetes mellitus," *The Lancet*, 1884, vol. i. pp. 338 and 384.—48. HÉDON, E. "Influence de la piqûre du plancher du quatrième ventricule chez les animaux rendus diabétiques par l'extirpation du pancréas," *Arch. de Phys.* 5me série, vol. vi. p. 269; "Sur la pathogénie du Diabète," *Ibid.* 1892, p. 247; "Greffe sous-cutanée du Pancréas," *Ibid.* 1892, p. 617.—49. HILDEBRANDT, H. "Ueber eine Wirkung des Piperazins und seinen Einfluss auf den experimentellen Diabetes," *Berlin. klin. Wochenschr.* 1894, No. 6.—50. HIRSCHBERG, J. "Diabetic Retinitis," *Deutsche med. Wochenschr.* 1890, p. 51.—51. HIRSCHFELD, F. "On a new clinical form of Diabetes," *Zeitschr. f. k. Med.* Bd. xix. Hefte 3 and 4; "Die Bedeutung der Acetonurie für die Prognose des Diabetes," *Deutsche med. Wochenschr.* 1893, No. 38.—52. KONIGMANN, G. "Ueber Magenthatigkeit bei Diabetes mellitus," *Deutsche med. Wochenschr.* 1890, No. 43.—53. HOPPE-SEYLER, G. "Beitrag zur Kenntniss der Beziehungen der Erkrankung des Pankreas und seiner Gefässe zum Diabetes mellitus," *Deutsches Arch. f. k. Med.* Bd. xxxii. Hefte 1 and 2, p. 171.—54. JAKSCH, R. v. *Clinical Diagnosis.* Eng. trans. London, 1890.—55. KAPOSI, M. "Ueber besondere Formen von Hauterkrankung bei Diabetikern; dermatosis diabetica," *Wiener med. Wochenschr.* Jan. 1884.—56. KAUFMANN, M. "Mécanisme du Diabète pancréatique," *Le Progrès Méd* 1894, vol. i. pp. 191, 223, 285; "On the Pathogeny of Diabetes mellitus," *The Medical Week*, 1895, vol. iii. p. 73.—57. KENNEDY, R. A. "Nitroglycerine in Diabetes," *Canada Med. Record*, Jan. 1888.—58. KINGSBURY, G. C. "Jambul seeds in Diabetes," *Brit. Med. Journ.* 1887, vol. i. p. 617.—59. KULZ, E. "Beiträge zur Kenntniss der activen β-Oxybuttersäure," *Zeitschr. f. Biol.* Bd. xxxiii. 1886, p. 329.—60. KUSSMAUL, A. "Zur Lehre vom Diabetes mellitus," *Deutsches Archiv f. k. Med.* 1874.—61. LANCEREAUX, E. "Notes et réflexions sur un cas de Diabète sucré avec altération du Pancréas," *Bull. de l'Acad. de méd.* 2e série, vol. vi. 1887, p. 46; "Le Diabète maigre et le Diabète gras," *L'Union Méd.* vol. xiii. 1880, p. 16.—62. LATHAM, P. W. "Pathological Relations between Diabetes, Gout, and Rheumatism," *Brit. Med. Journ.* 1885, vol. ii. p. 1053.—63. LEO, H. "Ueber die Ebstein'sche Theorie des Diabetes mellitus," *Centrlblt. f. k. Med.* 1892, No. 25; "Ueber die respir. Stoffwechsel bei Diabetes mellitus," *Zeitschr. f. k. Med.* Bd. xix. 1891, Suppl. p. 101.—64. LÉPINE, R. *Le ferment glycolytique.* Paris, 1891. "Étiologie et pathogénie du

Diabète sucré," *Revue de Méd.* Paris, 1894; "The Production of the Glycolytic Ferment," *The Med. Week*, 1895, p. 56; "A new Treatment of Diabetes," *The Med. Week*, 1895, p. 259. —65. LÉPINE, R., and MARTZ. "Sur le ferment glycolytique produit artificiellement aux dépens de la Diastase du malt ou du Pancreas," *Arch. de Méd. Exp.* vol. iii. 1895, p. 219.—66. LUSTIG, A. "Sugli effetti dell' Estirpazione del plesso celiaco," *Arch. per le Scienze Med.* 1889, No. 6.—67. M'AVOY, J. "Glycerinum Acidi Carbolici in Diabetes mellitus," *The Lancet*, 1885, vol. i. p. 736.—68. MACKENZIE, H. W. G. "The treatment of Diabetes mellitus by Pancreatic Juice," *Brit. Med. Journ.* 1893, vol. i. p. 63.—69. MARCHAL, DE CALVI. *Recherches sur les Accidents diabétiques et essai d'une théorie générale du Diabète.* Paris, 1864.—70. MAX EINHORN. "Die Gährungsprobe zum qualitativen Nachweise von Zucker im Harn," *Virchow's Archiv*, Bd. cii. p. 263.—71. MERING, VON, and MINKOWSKI. "Diabetes mellitus nach der Extirpation des Pancreas," *Arch. f. exp. Path.* Bd. xxvi.—72. MERING, J. VON. "Ueber Diabetes mellitus," *Zeitschr. f. klin. Med.* Bd. xiv.—73. MINKOWSKI, O. "Diabetes mellitus nach der Extirpation des Pankreas," *Arch. f. exp. Path.* Bd. xxxi.; "Ueber das Vorkommen von Oxybuttersäure im Harn bei Diabetes mellitus, ein Beitrag zur Lehre von Coma diabeticum," *Ibid.* Bd. xviii. 1884; "Pancreatic Diabetes," *Suppl. to Centrlblt. f. k. Med.* 1892.—74. MOLESCHOTT, J. "Iodoform gegen Diabetes mellitus," *Wien. med. Wochenschr.* Bd. xxxii. 1882.—75. MONCKTON, F. A. "Sulpho-carbolate of Soda in Diabetes mellitus," *Austral. Med. Gazette*, 1884.—76. MONIN. "Traitement du Diabète," *Le Progrès Méd.* 1894, vol. i. p. 265.—77. MORAT and DUFOUR. "Les Nerfs glyco-sécréteurs," *Arch. de Phys.* 5me série, vol. vi. p. 371.—78. MULLER, F. "Pneumaturia," *The Practitioner*, Jan. 1890.—79. MUNK, J. "Zur quantitativen Bestimmung des Zuckers und der sog. reducirenden Substanzen in Harn mittelst Fehling'scher Lösung," *Virchow's Archiv*, Bd. cv. p. 63.—80. OLIVER, T. "Cocaine in Diabetes," *The Lancet*, 1889, vol. ii. p. 735.—81. PATON, D. N. "On Hepatic Glycogenesis," *Phil. Trans.* vol. clxxxv. B. pp. 233-277; "A Contribution to the Study of the Influence of Fever on Hepatic Glycogenesis," *Edin. Hosp. Reports*, vol. ii.—82. PAVY, F. W. *The Physiology of the Carbohydrates.* London, 1894.—83. PEIPER, E. "Experimental Studies on the Results of Extirpation of the Cœliac Plexus," *München. med. Wochenschr.* April 29th, 1890.—84. PERCY, J. "Case of Diabetes mellitus," *Med. Gaz.* 1842-3, vol. i. p. 49.—85. PIATKOWSKI, M. "Ueber die therapeutische Wirkung des Benzosols bei der Zuckerharnruhr," *Wiener med. Wochenschr.* 1892, No. 51.—85*a*. POOLE. *Cookery for the Diabetic.*—86. PRYCE, T. DAVIES. "A certain Cutaneous Affection occurring in Diabetes," *The Lancet*, 1885, vol. i. p. 938; "On Diabetic Neuritis, with a Clinical and Pathological Description of three Cases of Diabetic Pseudo-tabes," *Brain*, 1893, p. 416.—87. QUANJER, J. A. "Use of the Bark of Syzygium Jambolanum in Diabetes," *Weekbl. v. k. Nederl. Tijdschr. v. Geneesk*, 1888, vol. i. p. 251.—88. REYNOLDS, E. S. "On the Treatment of Diabetic Coma," *Medical Chronicle*, 1891, p. 338.—89. ROLLO, J. *Cases of the Diabetes mellitus*, 2nd edition. London, 1798.—90. ROQUE DÉVIC and HUGUENENQ. "Du Coma Diabétique," *Revue de méd.* 1892, p. 995.—91. SANDMEYER, W. "Beitrag zur pathologischen Anatomie des Diabetes mellitus," *Deutsch. Arch. f. k. Med.* vol. l. 1893, p. 382.—92. SANSONI, L. *Riforma medica*, 1891, No. 2.—93. SAUNDBY, R. *Lectures on Diabetes*, p. 83; "The Middlemore Lectures on the Retinal Affections of Bright's Disease and Diabetes," *Birm. Med. Review*, 1892, vol. i.; "A Case of Diabetes treated by Lævulose," *Internat. Clinics*, vol. iv. series 3, 1894; The Bradshaw Lecture, 1890, *The Lancet*, 1890, vol. ii. p. 381.—94. SCHMITZ, R. "Kann der Diabetes mellitus übertragen werden?" *Berl. klin. Woch.* 1890; *Prognose und Therapie der Zuckerkrankheit*, Bonn, 1892.—95. SCUDAMORE, C. *A Treatise on the Nature and Cure of Gout.* London, 1816.—96. SÉE, GERMAIN. "Antipyrin in Diabetes mellitus," *Brit. Med. Journ.* 1889, vol. i. p. 1472.—97. SMITH, A. SHINGLETON. "Remarks on the Morbid Anatomy and Pathology of Diabetes," *British Medical Journal*, 1883.—98. SOLIS-COHEN, S. "Lævulose and Strontium Bromide in the Treatment of Diabetes mellitus, with three illustrative Cases," *Internat. Clinics*, vol. iv. series 3, 1894.—99. SQUIRE, B. "A Case of Diabetes benefited by Phosphorus," *Brit. Med. Journ.* 1889, vol. ii. p. 1216; 1890, vol. i. p. 293.—100. SYMPSON, E. M. "Salicylate of Sodium in the Treatment of Diabetes," *The Practitioner*, 1891, No. 8.—101. TEISSIER. "Sur le traitement du Diabète sucré," *Bull. de Thérap.* vol. cviii. p. 332.—102. TYSON, J. "Diabetes mellitus," *Internat. Clinics*, vol. iv. 3rd series, Edin. and Lond. 1895.—103. VALENTINE, D. "Le Créosote contre le Diabète," *L'Osservatore*, Jan. 5, 1889.—104. VILLEMIN. "Traitement du Diabète aigu," *Bull. de l'Acad. des Sciences, Arch. Gén. de Méd.* 1887, vol. i. p. 364.—105. WEINTRAUD and LAVES. "Ueber den respira-

torischen Stoffwechsel im Diabetes mellitus," *Ztschr. f. phys. Chemie*, Bd. xix. 6, 1894, p. 574.—106. WEST, S. "The Treatment of Diabetes mellitus by Uranium nitrate," *The Med. Week*, 1895, p. 374.—107. WEYL and APT. "Ueber den Fettgehalt kranker Organe," *Virchow's Archiv*, Bd. xcv. p. 351.—108. WHITE, W. HALE. "On the Effect of giving Lævulose and Inulin to Patients suffering from Diabetes mellitus," *Guy's Hospital Reports*, vol. l. p. 132.—109. WILKS, S. "Cases of Diabetes treated with Nux vomica and Mineral Acids," *Med. Times and Gaz.* 1884, vol. i. p. 320.—110. WILLIAMSON, R. T. "Changes in the Posterior Columns of the Spinal Cord in Diabetes mellitus," *Brit. Med. Journ.* 1894, vol. i. p. 398; "The Condition of the Pancreas in Fourteen consecutive Cases of Diabetes mellitus," *The Lancet*, 1894, vol. i. p. 927.—111. WILLIS, T. *Pharmaceutice rationalis sive diatriba de medicamentorum operationibus in Humano Corpore.* Oxonio, e theatro Sheldonio, prostant apud Ric. Davis, 1679, Part I. sect. iv. cap. iii. p. 85.—112. WOLPE, H. "Untersuchungen über die Oxybuttersäure des diabetischen Harns," *Arch. f. exp. Path. u. Pharm.* Bd. xxi. 1886, p. 140.—113. ZALESKI, A. "Zur Pathologie der Zuckerharnruhr (Diabetes mellitus) und zur Eisenfrage," *Virchow's Archiv*, Bd. civ. p. 91.

R. S.

DIABETES INSIPIDUS

Classification.—Under the names "Diabetes insipidus," "Diuresis," "Polyuria," "Polydipsia," writers have described a certain morbid condition of the system, characterised by excessive and persistent discharge of urine of low specific gravity which, unless in exceptional cases, contains neither sugar nor albumin. Most authors apply any one of the above names to denote this urinary superflux, without reference to the quantitative relationship that may exist in individual cases between the urinary water and solids. Others, of whom Willis seems to have been the first, have attempted to form a classification on this basis. Thus Willis divided cases of diabetes insipidus into three groups: (i.) those attended with an excessive discharge of aqueous urine, in which the solid matters are hardly affected—*hydruria;* (ii.) those attended with a copious discharge of urine with a deficiency of urea—*anazoturia;* (iii.) those in which the excessive discharge of urine was accompanied by a superabundance of urea—*azoturia.* Parkes also supported the view that diabetes insipidus is to be found under three different conditions: (i.) In cases where there is no increase or decrease of tissue metamorphosis; (ii.) in cases where there is a decided decrease of tissue metamorphosis; (iii.) in cases where there is evidence of increased tissue metamorphosis, as shown by the increase of some of the urinary solids. To this latter class of cases Dr. Parkes thought the term polyuria more apt than that of azoturia, for the latter only expresses the fact of the increase of urea; whereas in the cases quoted by him the fixed salts—chlorides, sulphates, and phosphates—were usually present in abnormal quantities. Again, the term *baruria* was introduced by the late Dr. Fuller of St. George's Hospital to describe certain forms of dyspepsia associated with excess of urea in the urine, in which only the solid matter of the urine is increased, not the water. Lastly, Dr. Tessier of Lyons has recorded a series of cases closely resembling saccharine diabetes in the increased

discharge of urine, the thirst, the neuralgic and rheumatic pains, the wasting, and secondary lung complications, but in which the urine contained no trace of sugar; a constant phenomenon in these cases was a very considerable increase in the quantity of phosphoric acid excreted, whilst the urea was not proportionally increased, a fact which Dr. Tessier thinks serves to distinguish them from cases of azoturia; this distinction he marks by naming the group *phosphatic diabetes.*

Though many writers still reserve the term "diabetes insipidus" for those cases in which a superflux of the urinary water is seen without any increase of the urinary solids, still it must be admitted that many cases present both characters. It therefore seems, so far as our present knowledge of these conditions is concerned, that this increased excretion of urinary water and of normal urinary solids respectively should be referred to two classes: (A) that in which the aqueous superflux is most marked —*hydruria;* (B) that in which the drain of one or more of the solid constituents of the urine is persistent—*polyuria:* this classification will be used in this article.

A. HYDRURIA.—**Definition.**—Hydruria is that form of "diabetes insipidus" which is characterised by a superabundant discharge of aqueous urine without any proportionate increase of the normal solid matters of the urine, and without the presence of such abnormal products as sugar or albumin, unless it be in the way of complication or towards the end of life.

Ætiology.—Hydruria, according to hospital statistics, is a somewhat rare disease. According to the register of the London Hospital from 1876 to 1895, only eight cases were admitted during that period, of whom four were males and four females. The average age was 36 years; the youngest patient was 23, the oldest 55 years of age. Taking, however, out-patient experience, I find that, during fifteen years, twenty cases have been under my care, which would represent say about a sixth of the whole number of such cases attending that department of the hospital. To these may be added nine cases observed in private, and thirty-two cases obtained from trustworthy sources. The following table shows the average age of these cases at each decimal period, and the proportion of males and females:—

Age.	Males.	Females.	Total.
1 to 10 years . .	12	7	19
10 ,, 20 ,, . .	6	3	9
20 ,, 30 ,, . .	6	4	10
30 ,, 40 ,, . .	15	7	22
40 ,, 50 ,, . .	4	2	6
50 ,, 60 ,, . .	0	1	1
60 ,, 70 ,, . .	1	1	2
	44	25	69

It will be seen from the above that the two periods of life in which the disease is most frequent are those of early childhood and early middle age, and that the proportion of male to female cases is about two to one up to the age of 50; after that age the number of cases is too few for a fair estimation. This table accords with the view expressed by other authors, that the disease affects males in the proportion of "two or three to one," and that it is chiefly a disease of middle life; but I think it emphasises the fact that the disease is nearly as prevalent during the first decade of life. The youngest patient under observation was a male child at 2, the oldest a man 69. This last patient, a fine, well-built farmer, said he had always been a "leaky" subject, passing much more urine than he should. Of late, during the cold weather of February of 1895, the diuresis had much increased, the urine on measurement amounting to six pints per diem, with a specific gravity of 1·005.

Trousseau has remarked that it is not unusual to find patients suffering from "diabetes insipidus" whose parents were either glycosuric or albuminuric. This is especially the case with young children; but, on examining a considerable number of cases at all ages, I find that heredity has a wider range of constitutional dyscrasia than these two varieties. Thus of twenty-seven patients whose family history I was able to trace, in twelve either one or both parents were tubercular, and seven presented a distinct history of pulmonary phthisis. In five cases there was undoubted evidence of inherited syphilis, three of these were children; in three the parents had suffered from gout, two of these were adult patients; in two, one parent of each had died from heart disease with a history of rheumatism; two gave a family history of saccharine diabetes; in three, either the father or the mother had died of Bright's disease; but in at least two of these last cases the albuminuria may have been due to lardaceous degeneration consequent on phthisis.

Among the remoter causes among children malnutrition stands first and foremost. It is the children of the poorer districts of London which furnish the bulk of the younger sufferers from this disorder, among whom such constitutional taints as tuberculosis or inherited syphilis are found, combined with ill-feeding and neglect. Certainly the disease is not common among children of the easier classes. Of thirty-four cases in which I have been able to investigate the personal history, eight were directly preceded by neglect, improper feeding, and a constitutional taint. Three cases seemed attributable to protracted worry and anxiety, four to alcoholism, three to acquired syphilis. In four cases the disorder was developed after influenza; three of these patients recovered. Three cases followed exposure to cold: one patient had fallen into the water, and sat a long time afterwards in wet clothes; another had drunk cold water when heated; and another had been much exposed to the great cold of the February of 1895. In one case the disease was associated with heart disease consequent on rheumatic fever. Three cases followed injury to the head; in these the diuresis after a time gradu-

ally declined. In three cases a tumour of the brain was found on autopsy. In two cases I found the disorder associated with aneurysm of the aorta, a fact which is interesting in connection with Dr. Dickinson's case of "diabetes insipidus" associated with an abdominal tumour involving the solar plexus.

To sum up the ætiological factors, we may then say that hydruria is a disorder that mainly affects early childhood and middle adult life; that males suffer in proportion of two to one of females; that hereditary predisposing factors mainly manifest themselves on the lines of tuberculosis, inherited syphilis, and gout; and that among children the parents have often been either glycosuric or albuminuric. Of the more immediate causes anything tending to depress the nervous system may play its part; among such events are malnutrition and neglect, long-continued worry, heavy drinking, syphilis, depressing illness such as influenza, and exposure to cold. Among the more direct interferences with the nervous system are injuries to the head, tumours of the brain, pons, or medulla, and tumours pressing on the thoracic and abdominal nerve ganglia, which probably agree in disturbing the vaso-motor governance of the renal vessels.

Symptoms.—The two symptoms of the hydruric form of "diabetes insipidus," which alone are characteristic of the disease, are a profusion of urine without sugar, of low specific gravity, and an intense thirst.

Urine.—In well-marked cases the urine may be increased tenfold—from two and a half pints to over twenty pints. The highest quantity I have myself observed was sixteen pints in the twenty-four hours. The diuresis may certainly be more extreme than in saccharine diabetes. Up to a certain stage of the malady the amount of urine passed is in excess of the total of fluids ingested, the excess being made up of the withdrawal of fluid from the tissues and from the food. Subsequently, as the appetite fails and the tissues become dehydrated, this disproportion is lost, and the total amount of urine falls below the aggregate of the fluids taken in. The colour of the urine is variously described as of a pale yellow or greenish tint. In characteristic cases, when fresh and examined through a tall glass cylinder, it has often a pale bluish tint, like that of the Sicilian aquamarine. When first passed it is faintly acid, or perhaps more frequently neutral; but it soon becomes turbid from the deposition of epithelial cells, and from ammoniacal decomposition causing precipitation of phosphates. The specific gravity is always low, but the statement that it has been known to fall as low as distilled water is founded on error of observation; the specific gravity must be estimated when the urine, freshly passed from the bladder, has a temperature of about 90° F., and not when it has cooled to the standard temperature of 60° F. The difference of 30° F. would represent something more than four degrees of specific gravity; a specimen of urine freshly passed from the bladder at a temperature about 90° F., and registering on the urinometer a specific gravity of 1·000 to 1·001, would if again estimated at 60° F.—the standard temperature—register a specific gravity of from 1·004 to

1·005. The lowest specific gravity taken at 60° F. was, in a child, 1·002; in an adult 1·003. Even figures so low as these, when the amount of diuresis is taken into account, show an increase in the amount of urinary solids above the normal; such an increase, however, does not denote an increased metamorphosis of tissue, but rather a washing out of the tissues by the excessive drainage, and an excessive ingestion of food. At the most the solid matters are increased by a fourth to one-third. As may naturally be supposed, uric acid is not deposited from such dilute urines; but it not infrequently appears when from any cause the diuresis is very greatly diminished. The most usual deposits are those of oxalates and calcium phosphates. Glucose, though no feature of the disease, may occasionally appear in small quantities; or it may appear towards the end of the case, when indeed it may merge into saccharine diabetes. Inosite or muscle sugar is not an infrequent constituent; in some cases I have observed it in considerable quantities. Albumin may be present in small quantities throughout, but it usually makes its appearance towards the end of the case as a part of the usual dyscrasia.

The intense *thirst* is as well marked a feature as the diuresis, if not more so. Clinical records tell us of heroic potations. Trousseau has recorded the case of a patient who consumed fifty pints of fluid daily; his urinary secretion was about fifty-six pints. The same patient, on one occasion when the amount of drink had been restricted, had drunk the contents of his chamber-vessel! There is no doubt that the thirst in cases of "polydipsia" exceeds that of saccharine diabetes; moreover, at first the urinary secretion continues in excess of the amount ingested, showing that both the tissues and the food are drawn upon to supply the aqueous drain: as the disease advances the balance becomes more equal, and as dehydration of the tissues increases more fluid is required to make up for the body loss; thus after a time the fluid ingested is more than the urine excreted.

Among other symptoms noticed in the progress of the disorder are *disturbances of innervation*. In the early stages the patient often complains of severe and racking pains in the lumbar region shooting down into the thighs. In one acute case, admitted into the Seamen's Hospital in 1876, the patient was in a stupid, semi-comatose condition, in which he remained for several days; he then gradually improved, though he was still passing large quantities of water. In another case, in an out-patient who had attended for several months at a large London hospital for dyspepsia, the disorder set in suddenly with violent pain in the head and sudden blindness: the urinary secretion rose to thirteen pints a day, and continued to increase till the time of his death, which took place shortly afterwards. Among the minor disturbances of innervation are the frequent tendency to headaches, mostly in the occipital region; spasmodic affections of the gastro-intestinal tract, such as choking sensations, hiccough, and irregular action of the bowels; itching of the skin and other disturbances of the sensory functions, and certain spasms of particular groups of muscles. Impotence is a marked symptom throughout. Amblyopia without any

special lesion is frequent. Towards the end insomnia is usually a very distressing symptom; it often passes abruptly into coma, preceded or followed by convulsions. Special nerves are not paralysed unless there be some corresponding cerebral lesion; nor are ophthalmoscopic changes associated with the disorder unless it be in connection with some concurrent dyscrasia, as in the case of the blind patient mentioned above in whom neuro-retinitis was observed together with fulness of the retinal vessels: in this instance the section revealed the existence of a small gumma situated in the middle line under the floor of the third ventricle, and some softening of the brain substance around it. As in saccharine diabetes, the reflexes disappear at an early period, though I have met with cases in which they have still been elicited for some considerable period.

Nutrition.—Digestion is usually well performed, and this in spite of the enormous quantity of food that is often ingested in the early stage of the disease. The appetite, so long as the patient maintains his general health, is often voracious; and when we remember the large quantity of water required to supply the drain from the body, this is not to be marvelled at. Trousseau has recorded some of the wonderful performances of these sufferers, so that I need not emphasise them further; but I may testify that in many of my own cases their exploits have been no less heroic. As digestion is often performed with regularity, this "bulimia" may give rise to no inconvenience; but in cases that run a prolonged course, as the health fails the appetite flags, and is succeeded by disrelish for food, which increases until complete anorexia is reached. By this time the disorder has made considerable advance; the tongue has become red and glazed, the mouth and throat are covered with sticky mucus; the bowels, which before were obstinately constipated, now alternate between that condition and severe attacks of diarrhœa, which often become uncontrolable and hasten the patient to his end. Generally one of the troubles is a persistent flatulence which no plan of diet or medicament seems able to relieve.

Emaciation sets in very early; but so long as the appetite is maintained it does not progress so rapidly as towards the later stage, when anorexia becomes a prominent symptom. Then the muscles rapidly waste, and the skin becomes wrinkled and assumes a dry, withered appearance. Owing to the extreme muscular weakness even the slightest exercise becomes impossible, and the patient either takes to his bed, or rests all day on chair or sofa.

The *temperature* is always subnormal, unless it be raised for a time by pyrexial affection; in this case it has been noticed that the abundant discharge of water falls considerably. The pulse is always feeble and easily compressible, with no sign of high pressure.

Among the symptoms that close the case, *simple exhaustion*, followed by *drowsiness* passing into *coma*, is perhaps the most frequent in chronic cases among adults; this exhaustion is often precipitated by a severe and uncontrollable attack of diarrhœa. A form of low congestive pneumonia

often carries off the patient already reduced by the excessive drain. In those who have a tubercular diathesis pulmonary consumption sets in early; in young subjects convulsive seizures and coma often announce the end.

Diagnosis.—In well-marked cases of "hydruria" there is little difficulty in recognising the disorder by the persistent discharge of large quantities of aqueous urine of low specific gravity, the intense thirst, and the progressive emaciation.

It is distinguished from saccharine diabetes by the absence of sugar from the urine and the low specific gravity. It certainly happens, however, in "diabetes insipidus" that small quantities of sugar appear in the urine; but in my experience it is always transitory, and the specific gravity is never high. In one case, when it first came under observation, the patient was passing six pints of urine; the specific gravity was 1·010, and it contained 4 grains of sugar to the ounce. Under moderately restricted diet and opiates the sugar entirely disappeared, and it has not reappeared since—a period of six years.

The condition known as anazoturia, or, as Sir Andrew Clarke named it, "renal inadequacy"—a condition in which the solids of the urine, chiefly the urea, are greatly diminished—may be taken for "hydruria"; but in that disease, if such it be, there is no absolute alteration of the urinary solids, and the amount of water discharged in diabetes insipidus far exceeds anything met with in "renal inadequacy": in these latter cases, although the specific gravity, in the absence of the urinary solids, is low, the urinary water often does not exceed the normal, or does so only to a slight degree. Most cases of so-called renal inadequacy, that is, of diminished excretion of solid matters, that have come under my observation have proved to be early stages of granular kidney.

The copious discharges of urine of low specific gravity by hysterical patients may for a time mislead the physician. But the general character of the patient, the usually intermittent recurrence of these discharges, and their amenability to anti-hysteric remedies, are sufficient to protect us from a false interpretation of the symptoms.

The discharges of aqueous urine from large cysts in the kidney may be misleading at first. Thus a child was brought to my out-patient room with a statement that, on the previous day, it had passed quite two pints of pale urine and was still passing more than it should do. Careful examination elicited nothing wrong; but on its next visit to the hospital I discovered a distinct tumour in the left flank which on pressure slowly disappeared; whereupon the child discharged more than a pint of urine by the bladder. The case was evidently one of a hydronephrosis, which, on reaching a certain point of distension, discharged itself by syphon action. The intermittency of the discharge, and the absolute deficiency of solid matter, ought to prevent our being misled after the first observation. In cystic disease of the young the urine is often of a pale, limpid character, of rather low specific gravity, usually albuminous, but not largely increased in amount. The detection

of the enlarged cystic organs usually determines the diagnosis; otherwise the absolute decrease of solid matters, while the excess of the urinary water, if any, is moderate, will settle the matter. Imposture is not readily detected, as it is only necessary to add the requisite amount of water to obtain the characteristic urine; but close observation will detect the cheat: indeed the patient could not suffer from the supposed diuresis without experiencing ere long the horrors of the water torture of the Holy Inquisition.

Frequency of micturition may sometimes be taken at first sight for increased discharge of urine. In the incontinence of adults evidences of obstruction of the outflow and the character of the urine itself are sufficient for diagnosis. The nocturnal incontinence of children is easily recognised; but a difficulty may arise in those somewhat rare cases in which the incontinence is diurnal as well as nocturnal: even here, however, a slight attention to the facts of the case will soon determine its nature. The act is almost unconscious, the child wetting itself when in school or at play without seeming aware of any mishap; the urine is of usual, or sometimes of increased specific gravity, and careful observation will show that the quantity passed in the twenty-four hours is not excessive.

Prognosis.—The progress of cases of "diabetes insipidus" is very variable—sometimes they run an acute course from beginning to end; sometimes they persist for a number of years without any visible deterioration of health beyond a feeling of weakness and general malaise. The most rapid case under my own observation was that of a man already afflicted with a syphilitic growth at the base of the brain; he first exhibited symptoms of hydruria one month before his death; and this symptom gradually increased, till the week before his death he was passing over thirteen pints. The most protracted case was that of a patient who first appeared in the out-patient department of the London Hospital, October 1880, and who has attended off and on since then till the present time. At first he attended for "phosphaturia" without any marked increase in the amount of urine passed; but as soon as the tendency to eliminate an excess of phosphates passed off, the excretion of a dilute aqueous urine manifested itself, and for the last fourteen years he has passed from four to seven pints of urine daily with a specific gravity 1·008 to 1·004. During the past two years he has complained of odd feelings with partial loss of consciousness, but without convulsion —evidently attacks of petit mal.

Attacks that set in acutely generally follow some sudden shock; such as blows on the head, sudden chilling of the body when hot, excess of alcohol, or worry and anxiety. After injury to the head the disease seldom becomes pronounced, and gradually passes off, unless some permanent brain lesion ensue. In these cases glycosuria is generally noticed during the early part of the case, and after persisting for some time passes off, leaving the patient hydruric; this condition, however, seldom lasts more than two years, unless some secondary brain mischief have resulted from the injury. Sudden chilling of the body when heated

has also a tendency to induce the disease in an acute form. A young farm lad, who drank a quantity of cold well water when heated after mowing, began to pass large quantities of urine; when brought as an out-patient to the London Hospital, and his urine measured according to instructions, he was found to be passing four pints daily. He continued as an out-patient for quite two years without much impression being made on the urinary secretion; and during the last two months of his attendance he showed signs of mental deterioration. In the case of a lighterman who fell into the river on a cold winter day, when heated with rowing on his barge, the disease set in quite acutely, and after two years no definite improvement was made. In a case of prolonged exposure to cold weather, the patient was an elderly farmer, aged sixty-nine, who had never had an illness in his life, except that he had always been a "leaky subject"; his urine enormously and continuously increased in amount with the setting in of the prolonged frost during February 1895. According to his statement he was passing from seven to eight pints daily; no improvement has yet been made in his condition. Anxiety and overwork have not, perhaps, so immediate an effect, but still one sufficiently well marked for the patients themselves to refer to it. In the case of an old lady aged sixty-four, who had always led a life of ease and comfort, profuse diuresis followed attendance on a sick sister and grief for her death. In this case, now in progress for three years, beyond the insomnia caused by frequent micturition at night, which is best relieved by cannabis indica and ammonium bromide, no manifest disturbance of health has yet occurred. It is an interesting fact that the nocturnal excretion is in excess of the diurnal; on an average the proportion is as three to two pints.

When the disease begins insidiously, and runs a chronic course, the factors concerned are those which gradually undermine innervation, and especially the inhibiting function of the renal vaso-motor system. Such factors are chiefly the acquired syphilis of adults, the inherited disease in children, chronic alcoholism, general self-indulgence, and such constitutional diseases as struma, gout, and rheumatism. Such patients seem to drift imperceptibly into the "leaky" state, and rarely come under observation till a very profuse discharge is established. Among middle-aged adults of this class there is generally a history of antecedent genito-urinary trouble, such as nocturnal incontinence of urine in childhood and of spermatorrhœa at and after the age of puberty; as the quantity of water discharged increases, there is a gradual decline of virile power till it is quite lost. Should the disease run a chronic course no troublesome intercurrent symptoms, such as boils, carbuncles, or cataract, arise as in diabetes mellitus; nor unless the patient be distinctly tubercular do pulmonary troubles set in early. When they do it is generally towards the end of the case, and are then chiefly of a chronic nature—a kind of dry, slowly-advancing pneumonia beginning at the apex, and gradually spreading to the other lobes of the lungs. More usually the end comes with symptoms showing the consequences

of the protracted dehydration on the nervous system, such as tremor, spasms, giddiness, and minor epileptiform manifestations. In protracted cases of hydruria the final stage is often painfully prolonged, unless cut short by an attack of congestive pneumonia, of uncontrollable diarrhœa, or of coma preceded by convulsions. Owing to the extreme muscular weakness the patient is unable to leave his bed, and lies coiled up in it, for the most part in an apathetic state. The skin becomes withered and covered with brawny scales, the tongue is red and glazed, and the breath horribly fœtid. The ravenous appetite by this time has given way to an extreme disgust for food, and even the polydipsia is much diminished. The abdomen is distended by flatulence; the bowels are constipated and the motions scybalous; this condition, however, not infrequently alternates with severe attacks of diarrhœa. The emaciated patient succumbs to one or other of the affections above named, and dies in the last stage of marasmus. Finally, as Dr. Saundby has repeatedly urged, death may be due to the gradual destruction of the kidney described in the section on the pathology of this disease. This secondary wasting of the kidneys in not a few cases leads to death by uræmia. Diabetes insipidus then may run a long course with little danger to life, or even none; on the other hand, we see that it is attended by grave dangers in more than one direction.

All cases of diabetes insipidus, however, do not end thus fatally; for though the drain of water may be excessive for many years, the general health may continue good and the digestive functions be performed with regularity. In such cases "hydruria," as Trousseau has remarked, constitutes an infirmity rather than a disease. The patient already referred to, who first sought medical advice for this complaint when sixty-nine years of age, was evidently one of this class; for he stated that for as long a time as he could remember he had been troubled by the tendency to pass more urine than was natural, and had always been more or less thirsty. It was not until he was chilled during the intense frost in February of 1895 that this mild form of hydruria assumed a more aggressive attitude. Otherwise he appeared a robust man, likely to reach his eightieth year. Again, in established cases, acute exacerbations may arise and threaten life, and yet subside, leaving the patient in the same state as before. The patient in the Seamen's Hospital in 1876, already referred to, was in an extremely feeble and emaciated condition, and passing large quantities of urine. There was no history of syphilis or injury to the head, nor was there any sugar in the urine. He lay in bed for several days in a stupid, semi-comatose state, yet after a time he gradually brightened and improved in his general condition. The diuresis, however, was still excessive during his stay in hospital, and so continued after his discharge. From his emaciated condition, and with nothing else to account for the comatose condition when admitted, it must reasonably be supposed that he was hydruric some time before he came under observation at the hospital. The disease may also set in acutely, and with great severity, and yet speedily subside

under careful tending and rest; this was the case with a sailor admitted into the Seamen's Hospital, one of four, who had been exposed in an open boat for several days at sea, suffering great privations and anxiety. This man, when admitted, was passing considerable quantities of aqueous urine, which gradually diminished with rest in bed and full diet, without any special medical treatment: he was discharged perfectly recovered. Remissions of the disorder even in well-established cases are not infrequent. A gentleman, now for some years under my care, tells me his trouble is less urgent during the winter months, when he gets a couple of days' hunting a week, than it is during the summer. The diuresis during the latter season averages quite seven pints daily; during the winter, on the other hand, it often does not rise above three pints for days together. That the effect is due to the exercise—probably to the sweating—and not to the season is shown by the fact that the diuresis increases whenever hunting is interrupted by a long frost.

In the prognosis of any individual case the facts already mentioned must be borne in mind. In mild forms of the malady, that are an inconvenience rather than a disease, an abatement may be looked for under proper management; always remembering that such cases may become aggravated, especially if we have reason to suspect any latent tubercular or syphilitic taint. Cases that set in acutely from some direct shock to the nervous system are often the most hopeful; while those, on the other hand, that begin insidiously and progress gradually, are the least amenable to treatment. When, however, the patient has once entered the downward stage—marked by anorexia, profound emaciation, and great muscular debility—the end is generally approaching; and, although there may be some lingering, when that stage is reached the victim rarely survives six months. In children, especially in those of tubercular parents, the disease never runs a protracted course; from beginning to end the period rarely extends to two years, and is usually not more than a few months.

Pathology.—The pathology of diabetes insipidus is a want of inhibitory control of the vaso-motor renal nerves. There are no characteristic changes in the kidney. In cases of a long course changes in the renal texture are sometimes seen, but only such as are common to other diseases, and have had nothing to do with the urinary superflux. Dr. Saundby, however, thinks that a survey of recorded cases leads to the opinion that prolonged distension of the urinary passages by enormous quantities of urine during a number of years leads to a diseased state of these parts which cannot be regarded as a mere coincidence. It is interesting to trace the course of the nerves forming the renal plexus, as irritation from eccentric or distant sources may play a part in inhibiting the renal nerves. Thus, the nerves forming the renal plexus are derived chiefly from the solar plexus; as the right vagus and great and lesser splanchnics join the solar plexus, it is probable that branches of these nerves enter the kidney by way of the renal plexus. The splanchnics also send branches direct to the renal plexus; and the left vagus sends

some fibres to the left kidney. They contain medullated and non-medullated fibres; and Krause has traced the latter as far as the apices of the papillæ. Their mode of termination is unknown. Physiologically vaso-constrictor, vaso-dilator, and sensory fibres have been ascertained. The connection through the vagus brings us into range with the medulla oblongata, and with many organs susceptible of tubercular or syphilitic growths, or of sudden shock, such as chill. The solar plexus may propagate the effect of abdominal new growths or aneurysms. In long-standing cases the kidneys are mostly enlarged, especially about the cortex, to which may be added slight granulation of the surface if albumin were present in the later stages. Another common occurrence is dilatation of the pelvis, often so far as to resemble a cyst. Thus it has been suggested that there may be two forms of diabetes insipidus: one characterised by a normal or perhaps slight excess of urea, the other by a subnormal excretion of urea. The post-mortem evidence, however, is not yet sufficiently conclusive to decide this point. The fact of the diminution of urea may be due merely to the anorexia that accompanies the latter stages of the disease, whilst at the early period with a voracious appetite the urea is naturally increased. After a prolonged period of diuresis, it is easy to anticipate sacculation of the pelvis, which is a reservoir for the superfluity; since the patients, wearied by the frequent efforts at micturition, naturally try to retain their urine as long as possible. The cause of the great fall in the amount of urine, often to normal, on the accession of pyrexia is unexplained, unless it be by the immense consumption of water that occurs in acute febrile states. The same effect is observed in diabetes mellitus; and I have noticed it in ascites, in which after the patient had been tapped twice the belly spontaneously emptied itself after a pyrexia (broncho-pneumonia) lasting three weeks. Since these lines have been in print I have heard of the termination of a case of diabetes insipidus, which I think of sufficient interest to insert. A somewhat pinched-looking young lady, aged twenty-seven, but looking only about nineteen, was brought to me two years ago suffering from thirst and occasionally voracious appetite. Her urine contained no sugar, but she was evidently passing much more urine than the normal. I ordered valerian, bromide, and arsenic. As she suffered from occipital headaches the optic discs were examined, but nothing wrong was found. I did not see her again till October 1895, when her condition was much the same, her expression perhaps a little more pinched. She also complained of obstinate constipation, for which cascara was ordered. In January of 1896 her maid heard her groaning in the closet; her mother went to her, and found her exclaiming, "Oh my neck, it is bursting; I am going blind, blind!" A small and latent aneurysm had burst at the base of brain.

Treatment.—A great variety of drugs has been recommended for this disorder, each being in turn regarded as more efficacious than the others. Among the first to be mentioned is valerian, which owes its reputation to the strong advocacy of Trousseau, who used it in heroic doses; in one case the quantity of 10 grms. in the twenty-four hours

was progressively increased up to 30 grms. In large doses, but far less than these, valerian soon disorders the stomach, and creates such nausea that it cannot long be effectively administered. When doses of half an ounce to an ounce of tincture of valerian are given four times a day in a wine-glass of water, with some compound spirits of ammonia and chloric ether to disguise the nauseating taste, a very encouraging result often follows. But the effect is soon lost, and any attempt to increase the dose fails on account of the nausea. Attempts to give the drug in the form of extract enclosed in capsules have the same disadvantage.

Assafœtida has been used as an adjuvant, but it has no apparent influence on the diuresis, and only adds to the nauseating effect. On the other hand, I have found that from 5 to 10 drops of tincture of cannabis indica seem to counteract nauseating effects, and to have a quieting effect on the patient. In a case where the sleep was much disturbed by the frequent rising to micturate, the diuresis was diminished, and the insomnia consequently relieved, by a draught consisting of 6 drachms of tincture of valerian, 10 drops of tincture of cannabis indica, and 30 grains of bromide of ammonium, with sal volatile and chloric ether in a wine-glass of water on going to bed; half this quantity was also taken twice during the day-time. For some time this mixture diminished the diuresis by almost one-half. As soon as the drug loses its influence, or the patient becomes intolerant of it, it should be stopped at once, and resumed again after an interval.

Ergot is another drug that has a good repute, and deservedly so; I have found that it effects a reduction in the amount of urine when other drugs have failed. Yet here again its power is limited; for if long continued or given in large doses, symptoms of ergotism appear, and its administration has to be stopped. Thus a farm lad, who attended for a considerable time as an out-patient at the London Hospital, was at first placed on 10-drop doses of liquid extract of ergot thrice daily in an iron mixture. The average quantity of urine at that time was seven pints, which steadily fell to a little over three pints. The drug was administered for many months, and the patient gained in weight and improved in general health, but eventually signs of ergotism appeared.

Nitro-glycerine has also been employed with good results. In one case, a male, aged forty, admitted as in-patient to the London Hospital, passing a large quantity of urine, and at first treated with valerian and other remedies without much benefit, was placed on nitro-glycerine; at that time he was passing 12 pints of urine daily; after four days the urine was reduced to 2½ pints, and the patient was discharged with the report "improved."

These are the chief remedies that have been tried and recognised as useful; some may be more effectual in one form of the disorder, others in another. But no such distinctions can be made till the history of a considerable number of cases comes under review. We do not need so much an enumeration of indiscriminate instances in which one drug is useful, as the actual condition of the individual, especially as regards

his family and personal history, and a notification of the reasons for the employment of a certain drug, and the probable causes of its success or failure.

Among other drugs that have been employed may be mentioned opium and its preparations, strychnine, quinine, belladonna, nitrate of silver, salts of zinc, iron, and arsenic. These are rather useful adjuvants than primary remedies. Opium and its preparations, unlike their action in diabetes mellitus, have but slight effect in the insipid form of the disease. This we can readily understand, since opium in the former instance checks diuresis by checking the formation of sugar in the system, and by diminishing the amount of this diuretic body passing through the kidneys; thus the beneficial effect of opium in reducing the diuresis in the saccharine disease is nearly always proportional to the diminished quantity of sugar excreted. In diabetes insipidus no such reduction in the superflux is observed, though some diminution does to a certain extent occur, which is no doubt due rather to a check in the metamorphosis of tissue and in dehydration than to direct action on the renal circulation. Whilst making this statement, it is only fair to record the case of an in-patient of the London Hospital, a woman aged thirty-eight, with a tubercular family history, who, while passing fifteen pints of urine daily, was placed on codeia; the amount of urine fell quickly to two pints, but as she died in hospital it is possible that the disease was near its termination: no autopsy seems to have been made, or some explanation of this unusual and energetic action of codeia might have appeared. The preparations of opium are very useful in advanced cases to relieve the insomnia, restlessness, and subjective nervous symptoms, and to check the advance of pulmonary symptoms. Quinine and strychnine are useful tonics—the latter being especially useful when there is much muscular weakness and convulsive twitching. Iron is always useful as an adjuvant, and combined with ergot, much increases the efficiency of the latter drug. The more soluble preparations are the most suitable and best tolerated. In the latter stages of this disease it is called for to check the profound marasmus and anæmia; especially when these are attended with extreme muscular debility and passive œdema. The salts of silver and zinc by their action on the central nervous system have proved beneficial when convulsions, twitchings, or symptoms of an epileptiform character set in. I have already stated that the valerianate of zinc has in some cases a decided power of diminishing diuresis.

Arsenic may always be relied on to improve the general condition in all stages of the disease. It relieves the excessive appetite in the early and the anorexia in the later stages: under its action the skin often loses its withered appearance, and the bowels become less constipated. When given in combination with some of the special agents already mentioned I have observed that it greatly increases their power.

In syphilitic or tubercular subjects specific remedies must also be employed: for the former, mercurials and iodide of potassium, but with caution; for the latter, iodide of iron and cod-liver oil. In a case, recorded

by Dr. Robertson, of diabetes insipidus dependent upon disease of the bulb, the only remedial measure that seemed to be of distinct use was voltaic electricity. The positive pole was applied to the back of the head and neck by a large electrode, and the negative, duly insulated to within a quarter of an inch of the point, was passed along the floor of the nostril till it rested on the cervical spine. The strength of the current was gradually increased from a half to five milliamperes, and its duration from one to five or six minutes each application. This was continued every second or third day for seven weeks, when the patient felt so well that he left the hospital to begin work on December 15, 1888. The urine, on admission 260 ounces and upward per diem, gradually diminished in quantity, the amount ranging from 78 to 88 ounces in the twenty-four hours during the last fortnight of his residence. The specific gravity had risen to 1010 or 1012. He was three times in hospital, and obtained the same relief by this means from the polyuria on each occasion, though the palsy due to the central disease persisted.

Symptoms arising from intercurrent diseases must be treated on general principles. The only incident which calls for attention here is that of constipation. This is caused by the dryness of the fæces from the drainage of water from the intestines, by the progressive weakness of the muscular walls, and by the excessive amount of food ingested. The accumulation occurs in the lower bowel, and impaction of fæces is apt to occur there unless care be taken to evacuate its contents. Irritant purgatives, especially those that affect the smaller intestines, should be avoided, as very slight irritation of the mucous membrane often causes severe diarrhœa of an aqueous character. Purgatives, when employed, should be those that act chiefly on the colon and rectum, such as colocynth and aloes combined with strychnine and belladonna. Resort may be had to enemas of simple water with a little castor oil added; or, if the obstruction be severe, a glycerine enema may be used. Flatulence, which is a very troublesome symptom, is not caused altogether by fermentative changes; the gas, which consists chiefly of nitrogen, may be the remnant of swallowed air, as is the "flatus" in the "passio hysterica." It often comes on suddenly, even after relief has only just previously been effected. A pill consisting of extract of valerian and assafœtida, with extract of cannabis indica, rubbed with oil of rue or cajuput, may give relief; to this a small quantity of some opiate may often be added with advantage. Massage over the abdomen should be employed daily, both for the purpose of prevention and of relief.

The *dietetic* as well as the hygienic treatment, though not so important in the insipid as in the saccharine form, still requires careful attention. First as to the quantity of fluid the patient should be allowed to consume in the twenty-four hours. In view of the almost insane craving ("hydromania") for fluid, and the enormous discharge of watery urine, the question has been considered whether the diuresis could be controlled by placing limits on the amount of fluid ingested. The experiment has been made, and in some cases the reduction in the amount of

fluid has been carried to a cruel extreme. No doubt restriction of the amount of fluid is followed by a diminution of the diuresis, and up to a certain point restraint may be imposed; but it must be remembered that at this point the tissues are drawn upon, and their dehydration will increase. In one case recorded by Trousseau, in which extreme restriction was resorted to, although the amount of urine was considerably decreased, the patient found "his strength decreasing, his sight failing, and his body wasting." The plan of procedure I generally adopt, when a patient first comes under observation, is to ascertain the daily quantity of fluid he is in the habit of taking and the daily diuresis. If these be nearly balanced I reduce the amount of fluid by two pints daily; if this be followed by a corresponding fall in the amount of urine passed, I make successive reductions of about one pint every third day in the amount of fluid taken. When no further reduction in the diuresis is effected, no further reduction in the amount of fluid ingested should be persisted in; otherwise the deficiency will be made up by an exhausting drain on the tissues. Thirst is greatly alleviated by allowing the patient to suck ice and by the use of acidulated drinks to which a little gum-arabic or glycerine is added. When the appetite fails it is a good rule to add some nutriment to the drink; thus milk and water, cocoa and milk, and light mutton broth may be taken. A tablespoonful of raw oatmeal stirred in a quart of water, and flavoured with a little lemon juice and grated lemon peel, forms a grateful drink. Tea and coffee increase the diuresis and should be avoided; the same objection applies to effervescing drinks.

With regard to the food there is not the objection to the use of carbohydrates which exists in saccharine diabetes, and they may be freely used; so may the hydrocarbons. With respect to proteid food, so long as the patient can digest and assimilate it, it may be given; but when anorexia occurs, and the process of metabolism is more or less arrested, then there is danger of overloading the bowels with undigested proteid material, of the absorption of toxic bodies, and of the formation in the tissues of toxins resulting from incomplete metabolism. Then the amount of proteid food should be limited, and given in as soluble and assimilable a form as possible. To aid reduction, the food should be minced; and some preparation of pepsin may advantageously be given with it. It is safe to place such a restraint on the voracious appetite as is not immediately followed by loss of weight or decline in strength; otherwise, if the patient enjoy his food and digest it well, no great severity need be exercised. By interposing small meals between the chief ones of the day much of the craving may be allayed; thus a plate of porridge, a basin of soup, or a poached egg is often sufficient to stay the stomach till the hour for the regular meal arrives. The amount of salt taken with food should be extremely small on account of its diuretic action; and of course all salted meats must be forbidden, unless bacon be permitted, and if so, none of the lean should be eaten, and care should be taken to select the fat parts (the back) from very mild-

cured flitches. It has been noted that some patients with this disease are very tolerant of alcohol; others, on the other hand, are extremely susceptible to it. Except in moderate quantities alcohol certainly increases diuresis, and it should therefore be taken as a dietetic only, and then not in the form of spirits; a glass of bitter beer or some claret and water is quite sufficient. As the patient becomes weaker a glass of port wine twice a day may be added to this.

Should sugar appear during the progress of the disorder, the dietetic treatment will be based on the principles laid down for diabetes mellitus.

The *hygienic* treatment is of great importance. Owing to the general relaxation of the nervous system, the patient should select a bracing place of residence, near the sea if possible; if away from the sea, at a place of some elevation and with a dry subsoil. As the body temperature is usually below the normal he should clothe warmly, wearing woollen both winter and summer; and he must avoid any sudden change of temperature. Exercise should be regular, but of a quiet character, and above all fatigue should be avoided. Walking, the gentle use of the bicycle, riding on horseback, and golf are the best forms of it. Rowing, football, or any exercise requiring sudden and violent movements, should be interdicted. In the later stages, when the patients, from muscular weakness, become unable to leave the house, efforts must be made to prevent their becoming bed-ridden. They should be made to rise daily, and encouraged to change their rooms, which, however, should be on the same floor. Massage to the limbs should be employed daily, but care must be taken not to overdo it. Care, too, must be taken of the skin, which from want of nutrition falls into a withered and atrophied state, and becomes extremely sensitive and painful to the touch, so much so that the patient cannot bear the weight of the bed-clothes; this intolerance adds to the insomnia and restlessness. The patient should be sponged daily with soap (carbolic) and water, and dried by the gentle friction of a soft Turkish towel.

B. Polyuria.—**Definition.**—Under the name polyuria three forms of urinary drain may be noticed: that of urea—"azoturia"; of the earthy phosphates—"phosphaturia," or, as Tessier and others name it, "phosphatic diabetes"; and "baruria," in which nearly all the solids of the urine seem to be increased together. To this form of urinary waste some continental writers have applied the name "demineralisation."

I. **Azoturia.**—This name was applied originally by Prout to a condition of urine characterised by the presence of urea in excess, though Prout did not distinguish between the increase of the urea and of the other solids. He pointed out, however, that it often preceded diabetes mellitus, but obtained little clue otherwise to its nature or pathology. Indeed, little advance has been made in this subject since his time. The affection is comparatively rare, and the cases are so chronic that they drift out of observation. No advance can be made until we distinguish between the single increase of urea, the increase of urea and

phosphates together, and the total increase of all the urinary solids. I have met with many more instances of the so-called "phosphatic diabetes" than of pure azoturia.

Azoturia, strictly defined, consists in an increase of the elimination of urea out of all proportion to the other solids. As urea is a diuretic there is often in this state an increase in the amount of water; this increase must often mask the disorder, if the specific gravity be alone relied upon, and no precise determination of the urea made. Thus, in a case lately before me, the daily urinary secretion amounted to just on 1800 c.c., or rather more than one-third of the normal, and the specific gravity to 1·023; the urea, however, was increased to 58 grammes, an increase of rather more than two-thirds calculated upon the man's age and weight. His diet had also to be taken into the calculation, but this condition will be referred to presently. The other urinary solids were in about normal quantity, except the earthy phosphates, which were slightly increased.

The disorder generally begins insidiously, with a feeling of languor; there is also an indescribable weariness, especially in loins and thighs. The tongue is usually pasty and foul, and the edges of it red and irritable. The stomach is evidently disordered, and intense cravings for food alternate with complete anorexia. If about this time the patient consults his medical adviser, and if his symptoms be properly interpreted, a little judicious management will probably prevent further mischief; and though the patient may still feel languid and tired, a visit to Carlsbad or Homburg, or better still, to some quiet grouse-shooting or deer-stalking resort in the Highlands of Scotland, will most likely put him right. If the opportunity be lost the disease will progress, the gastric disturbance will increase, and the tendency to anorexia and to thirst will become more pronounced. To the weariness in the loins succeeds actual pain, usually on both sides, though occasionally on one only; thus renal disease may be suspected, as I shall point out further on. Boils often appear; not the large boils of diabetes mellitus, but small ones the size of a hemp seed, with inflamed bases and yellow heads; these slowly develop, and frequently recrudesce. The skin becomes harsh, dry, and wrinkled from loss of adipose tissue, and the body-weight slowly but regularly declines. It is at this period that suspicion of diabetes mellitus arises, a more thorough examination of the urine is made, and the excess of urea and the absence of sugar are detected. Even in these extreme cases the patient may yet recover; otherwise the case may pass on, as Prout remarks, into diabetes mellitus, or the patient may perhaps die suddenly. An instance of that kind is vividly impressed on my mind, though it occurred nearly thirty years ago. An artisan in the Great Northern Works at Doncaster, who was regarded as a malingerer—as he frequently threw up a day's work and drew sick pay apparently on no substantial grounds—was at length discharged from the works, and his clubs were determined, if possible, to get rid of him likewise. He was brought to me, and I could discover nothing but a specific gravity of urine amounting regularly to 1·027-32, and crystals of urea which formed in it on the

addition of nitric acid: on reflection I thought it might be a case of azoturia. I therefore advised the officials to act cautiously in the matter. Ultimately one of the members took him up to London, and on my recommendation had him examined by three of the then leading urinary pathologists. Nothing except excess of urea could be detected, which showed, however, that he was passing nearly three times the normal amount of that substance. One of the physicians consulted took a grave view of the case; the other two took it rather lightly as a matter of diet and a change to the sea-side. Acting on this advice, he was sent by his clubs to a small fishing village near Hull. A few weeks after, without any manifest increase in his malaise, he went out for a stroll along the beach, came home, went up to bed, and when his wife went to call him for tea she found him dead. On section no morbid changes could be found in the body.

This disorder particularly affects male adults between the ages of forty and fifty-five. I have only once noticed it in a woman; that was at the "change," and it passed off as that epoch closed. Other writers on the subject mention the frequency of the malady among men. Of the pathology of the disorder we have no clear indication. It appears to be due to increased metamorphosis rather of tissue than of proteid food. Although restriction of the latter has a certain effect in reducing the amount of urea the result is never considerable; and a purely carbohydrate diet has very little influence in restraining the amount of urea. The best diet is a moderate mixed one, all stimulating substances being excluded.

The *causes* of azoturia are obscure; all my male cases were, with one exception, well-to-do men leading sedentary, busy, anxious lives, who for the most part lived neither wisely nor well. The other patient, the artisan I have mentioned, was evidently a man of constitutionally weak nervous organisation, and his trade was too heavy for him. The prognosis is favourable, especially in the early stages; even in advanced cases recovery will generally take place in two or three years unless diabetes or pulmonary complications ensue.

The *treatment* is to be directed to soothing the nervous system. The patient must rise late and go to bed early. The hours of work, if work be absolutely necessary, must be strictly limited; but if the patient can afford it and has strength enough for it, a cruise round the world is the best restorative. As an intermediate step a residence at Margate or a trip to the Highlands is to be advised. Of medicines I rely chiefly on preparations of opium and henbane; the latter seems to me to have a specially soothing effect. I usually prescribe a pill containing $\frac{1}{8}$ gr. morphia with 2 to 3 grains of extract of henbane. As a general tonic I give some mineral acid combined with a bitter infusion. The bowels should be kept well regulated. Every morning on rising the patient should have a "sea-salt" tepid shower bath, the temperature of which is to be regulated in accordance with the patient's power of resistance, and a good towelling must follow. Whilst care must be exercised not to let the temperature

fall too low, it is important that the patient should not be relaxed by too hot rooms, or clothes which are too heavy.

II. Phosphaturia. — This name is applied to three conditions of phosphatic urine: (*a*) that in which there may not be any excess of phosphoric acid, but in which the earthy phosphates are deposited simply on account of the alkalinity of the urine, usually the result of dyspepsia; (*b*) that in which ammonio-magnesian phosphate (triple phosphate) is deposited consequent on ammoniacal fermentation in disease of the urinary passages; (*c*) that in which an actual excess of the earthy phosphates is formed, which, if the urine be alkaline, are deposited in dense masses in the urine glass, or if it be acid, give the urine considerable density.

To prevent confusion between these three forms Tessier has proposed the name "phosphatic diabetes." In normal urine the proportion of alkaline phosphate to earthy phosphate is as 2 to 1 grms.; yet in the cases we are discussing the proportion is not infrequently as 2 to 5 grms. Tessier gives cases presenting even higher proportions, such as 9 grammes. In these cases the urea is sometimes increased, but not always; therefore it is that Dr. Tessier contends that some other name than azoturia should be applied to this condition. Of my cases, taking one with the other, the urea was one-third in excess, whilst the phosphoric acid in combination with the earthy basis was trebled. In another case the urea was nearly doubled, whilst the phosphoric acid combined with the earths was four times the normal amount. In only one case was the urea normal, and in it the earthy phosphates were exactly quintupled.

The following table shows the results of six cases in which exact and repeated analyses were made:—

	Age.	Weight.	Quantity.	Sp. Gr.	Phos. Acid.	Urea.
		st. lb.				
Case 1[1] . .	16	8 7	2900 c.c.	1·010	5·2	51·0
,, 2[2] . .	24	9 0	9600 c.c.	1·004	6·0	...
,, 3[1] . .	25	9 0	2300 c.c.	1·015	7·8	33·5
,, 4[1] . .	27	8 7	1520 c.c.	1·022	5·2	41·2
,, 5[1] . .	37	12 7	2020 c c.	1·018	7·6	48·3
,, 6[3] . .	20	9 0	3825 c.c.	1·016	6·7	87·2

In all the cases, about eighteen in number, that have come under my observation, in which the phosphoric acid has been considerably in excess of the urea, very severe and distressing constitutional symptoms have been associated with the excess. These symptoms vary considerably, but they are all characterised more or less by great nervous irritability, derangements of digestion, great emaciation, and severe aching pains in the loins and back especially affecting the pelvic viscera. The urine is copious, of medium specific gravity, either acid and clear, or sometimes

[1] These four cases seem to approach most closely Dr. Tessier's description.
[2] This case passed rapidly into one of diabetes insipidus.
[3] This case became one of diabetes mellitus.

alkaline and whey-coloured from deposited phosphates. The termination of the disease is variable. Of the cases I have been able to trace six have died of phthisis, which in two was associated with pleurisy; three passed into diabetes mellitus; and one into rapid diabetes insipidus.

Treatment.—The main indications are rest and promotion of general nutrition. To attain this end opium or codeia should be given in full doses as soon as the patient first comes under observation. When, however, the nervous system is quieted, and the rheumatic and neuralgic pains are abated, the opiate should be discontinued lest it interfere with digestion. General tonics, such as iron, phosphorus, quinine, nux vomica, hydrochloric acid, and cod-liver oil, should be perseveringly used. If there be a history of syphilis, iodide of potassium should be combined with these remedies. Warm baths, followed by tepid douches, give great relief to the neuralgic pains, and also soothe the nervous system.

The soluble phosphates may be administered, but their utility in these cases is questionable. There appears to be no lack of these constituents in the system; the difficulty seems rather to lie in some want of power in the tissues to retain them.

The food should be light and nutritious, and milk one of its chief constituents.

Alcohol should be avoided; even in small quantities it invariably increases the diuresis. The same may be said of coffee. The change to dry bracing air should be obtained if possible. The clothing should be warm, and the patient carefully guarded against cold, since in these cases a reduction of bodily temperature is always noted.

When in pursuance of these therapeutic and hygienic conditions the diuresis and excretion of phosphoric acid continue, though the general condition of the patient may temporarily improve, there is reason to fear that phthisis will supervene, or that the disease may assume the features of saccharine diabetes.

III. Baruria is the name applied by Dr. Fuller to certain urines which are characterised by a general increase throughout of their solid constituents, of one not more than of another, whilst the aqueous secretion remains tolerably constant. The specific gravity may run between 1·038 and 1·045, and at that it may remain for several days. If there were not an actual increase of the solid constituents one would attribute the heightened specific gravity to dyspepsia, or to a loss of water from some febrile condition; but in many cases I have found the urea, chlorides and sulphates increased by one-half. This state seems to be brought about by a quickened tissue metamorphosis, which may result in the formation of more acid toxins in the proteolytic cells which may dissolve out the alkaline bases too rapidly. However, the cases I have met have been in heavily overworked persons. These patients have complained of much the same symptoms as the polyuric and phosphaturic classes, and have been relieved by the same treatment, especially by the frequent use of the sea-salt tepid douches. Arsenic also seems to be of special value in this "demineralisation." A medical man residing in the Midlands who

suffered from this disorder drank freely of "La Bourboule" water, and said he could not do long without it.

CHARLES HENRY RALFE.

REFERENCES

1. *Lancet*, 1877.—2. ROBERTSON, ALEX. "Case of Diabetes Insipidus from Disease of the Fourth Ventricle," *Brit. Med. Journ.* Oct. 3, 1896.—3. SAUNDBY, R. *The Practitioner*, 1895, vol. liv. p. 39, and *Lancet*, 1897, vol. i. p. 432.

C. H. R.

LARDACEOUS DISEASE

General Pathology.—The condition to which this name is now generally applied has been also known as *waxy* and *amyloid* disease. The name *albuminoid* has been loosely applied both to this condition and to the somewhat similar, but essentially different, change in certain viscera which occurs in rickets; the name, then, may be dropped as equivocal. The lardaceous condition may be provisionally described as an infiltration; it is a special addition to the proper structure, the nature of which will be presently discussed. Chemically the new material is closely allied to some of the normal constituents of the body, but is distinguishable from them and readily identified by certain conspicuous reactions. The change is essentially general or constitutional; it is not local to any organ, but affects many organs and tissues simultaneously, though unequally. The change begins in the coats of the arteries, but soon extends beyond them, presenting itself rather as an incorporated addition to the normal organic elements than as a recognisable interstitial intrusion. In general terms it may be said that the solid organs when thus affected are increased in bulk, sometimes enormously; occasionally the enlargement is superseded by contraction, so that the affected organ may become, as sometimes in the case of the kidney, smaller than normal. The special alteration is generally distributed throughout the whole of the affected organ, though limited at first, and sometimes indeed altogether, to certain anatomical regions: in the liver it may be limited to the intermediate zones of the acini or territory of the hepatic artery; in the spleen to the Malpighian bodies; in the kidney to the Malpighian bodies and the straight vessels of the cones; in the mucous membranes to the arterioles. The arterioles are usually indeed the first seats of the change, which, however, extends beyond them and becomes somewhat general, involving morbid new growths, especially when of syphilitic origin; and sometimes even simple exudations such as urinary casts. The epithelial cells usually escape.

Chemistry of the Infiltration.—Before proceeding to any minute consideration of the affected organs and structures, I will say as much

as I think can be said safely with regard to the nature of the infiltrating material.

The ultimate composition of the lardaceous superaddition is that of the protein bodies. Organs displaying it in large amount have been subjected to ultimate analysis by Pavy and Odling, with the conclusive result that the morbid addition is nitrogenous and allied in composition to the protein bodies. The addition is not of the nature of starch or cholesterine, as has been supposed; it is allied to albumin and fibrin, though not identical with either of these bodies as presented in health, or obtained from bodies free from the disease in question. A remarkable and apparently an important property of this material, whether it be regarded as a new substance or as a modification of one properly belonging to the body, is its solubility in the fixed alkalies. The behaviour of the morbid material with liquor potassæ (and much the same may be said of liquor sodæ) is of great interest. When portions of lardaceous tissue are exposed to the action of this reagent, even in a dilute form, they lose at once and for ever their distinguishing reaction with iodine, and this before the tissue has been altered in any other respect discoverable with the naked eye or the microscope. The tissue so acted on may subsequently be saturated with acid—hydrochloric or acetic; but the power of reacting in the characteristic manner with iodine is not restored. Either the material to which the reaction is due has been dissolved out of the tissue, or else it has been so modified by the alkali as permanently to lose its special distinction. Something certainly is dissolved out, for a bulky precipitate can be obtained from the liquor potassæ by the addition of an acid. A very similar but not so bulky a precipitate can be obtained from the healthy tissue by a parallel process. The material thus dissolved out of the lardaceous tissue readily absorbs or unites with iodine, as does that obtained from healthy tissue; but it does not appear that the iodine reaction is decidedly different in the two cases. The lardaceous precipitate takes a fawn colour, as does the precipitate yielded by the healthy organ; the distinguishing iodine reaction does not survive the solvent process.

The matter dissolved out of the liver (which affords the best material for experiment) by potash in the manner described was found by Dr. Marcet to contain nearly the same proportion of nitrogen as belongs to albumin. The substance so extracted yielded on three analyses an average of 13·7 per cent of nitrogen, the percentage of albumin being 15·7. The morbid material so obtained could scarcely be perfectly pure, but nevertheless the resemblance to albumin is significant. But however the material may resemble albumin in ultimate composition, it differs from it in some important points: one of these is its solubility in water. Albumin, such as belongs to the healthy body, is thus soluble; the lardaceous matter, or at least as much of it as can be detected by iodine, is insoluble in water. The tissue may be macerated in water without losing its distinguishing reaction until it is destroyed by decomposition. Another and apparently a very important difference between lardaceous

and healthy tissue lies in the deficiency of potash in the lardaceous. I arrived at this fact as the result of incineration of the lardaceous and the corresponding healthy tissues, and Dr. Dupré, operating in a different manner, has confirmed the observation. I annex the results I have referred to in a condensed form :—

Mineral Constituents of the Liver and Spleen under Lardaceous Disease and other Conditions. The amounts are given as percentages, and as the means of the number of estimations stated.

Organ.	Observer.	Number of Estimations.	Salts of the Alkalies.	Salts of the Earths.	Potash.	Soda.	Phosphoric Acid.	Sulphuric Acid.	Chlorine.
Liver, healthy .	Dickinson	7	1·00	·057	·209	·196	...	...	...
,, lardaceous	,,	8	·739	·177	·169	·155	...	...	...
Spleen, healthy .	,,	3	1·07	·179	·307	·172	·118	·215	·096
,, lardaceous	,,	5	·799	·234	·196	·191	·164	·051	·098
,, rickety .	,,	1	·989	·095	·283	·196	·055	·025	...
Liver, healthy .	Dupré	3	...	.	·283	·948	·329	...	·150
,, lardaceous	,,	3	...	...	·131	·190	·198	...	·166

It appears that the lardaceous organs, both liver and spleen, are deficient in potash while they superabound in the earthy salts. It is of interest to contrast the lardaceous change with that of rickets, wherein, so far as a single instance can be relied upon, the opposite condition exists ; the diminution in rickets falls upon the earthy salts, not on those of the alkalies (2, 3). Thus it appears that the lardaceous element (or that part of the tissue which gives the distinguishing reaction with iodine) is something closely allied to, but in some respects different from the constituents of the healthy body. It is something superadded, for it entails increase of bulk, and can be readily removed—though in the process of removal by alkali it loses its characteristic reaction. It may be presumed that it is deposited in the place of the normal material as the result of some modification of the process of nutrition, of some chemical change—one probably small in itself, however important in its consequences.

It is well known that the reaction of the lardaceous tissue with iodine was discovered by Professor Virchow in searching for starch, on the hypothesis that products similar to those belonging to the vegetable kingdom might under certain circumstances of disease present themselves in the animal body. Starch is indeed abundantly present in the human body as belonging to the healthy process of assimilation ; but we do not know it as the result of disease, and it certainly has nothing to do with the "amyloid degeneration" of the German pathologist.

The lardaceous condition can be distinguished by several colour

reactions; some of the anilin colours and indigo act upon it differently than upon healthy tissue, but the iodine test is the readiest, and in some respects the most interesting. If a solution of iodine be brought into contact with the affected tissue that part takes a specially deep stain, and holds it longer than the unaffected intermediate structures. The colour is that of iodine itself, such as might be supposed to result did the affected tissues absorb the reagent more freely than the unaffected. For the purpose of the test the iodine may be dissolved in pure water, in spirit, or in a solution of iodide of potassium. To obtain clearness of result and avoid complications the most satisfactory solvent for the iodine is distilled water, by which it is dissolved slightly but appreciably. The water should be kept charged with iodine by the presence of an excess of this substance, and the tissue long suspended in the nearly colourless though concentrated solution. The water will gradually transfer the iodine to the tissue and produce the reaction simply and emphatically. The beauty of the test, as thus applied, is due to the deep coloration of the lardaceous parts, which is as intense as if a strongly coloured solution were used; while the non-lardaceous parts, being simply wetted with a virtually colourless liquid, display in contrast their normal colour. Solutions in spirit or iodide of potassium are prompt in action and suited for common use. Whether the solution be in iodide of potassium or spirit the further addition of sulphuric acid to the lardaceous tissue produces a change which has attracted much attention. The lardaceous tissue stained brown with the iodine becomes black, or nearly so, on the addition of the sulphuric acid. This colour was formerly described as blue, and was supposed to indicate the presence of starch. It is no longer necessary to insist that the blue is a delusion and the starch non-existent. The change of colour is due to the action of the acid upon the reagent, not upon the tissue; and it turns upon the fact that a solution of iodine in either iodide of potassium or spirit undergoes precipitation on the addition of sulphuric acid. This is easily shown by placing a little of either of these solutions on a porcelain surface, and superadding sulphuric acid; the precipitation and consequent darkening are sufficiently apparent. A similar change occurs if lardaceous tissue be stained by one of these solutions and the same acid applied. The change of colour does not appear, then, to be due to the tissue, but only to the mutual action of the reagents. The lardaceous tissue absorbs the iodine solutions abundantly, and displays the change in a corresponding manner. If the experiment be made upon lardaceous tissue which has derived the iodine stain from a pure watery solution, so that there is nothing involved which can be decomposed, no change of colour is produced by the acid.

Assuming, as we fairly may, that the exceptional coloration of lardaceous tissue by iodine is due to a special affinity between the tissue and the test, or a special power on the part of the tissue of absorbing the test, it becomes of interest to look at the relations of iodine to the constituents of the body under various conditions. A solution of iodine in

iodide of potash gives a large precipitate with a solution of albumin—taking, for example, the serum of ascites, which is alkaline and may be taken to represent the usual condition of albumin in the body. If the bulky chocolate-coloured precipitate thus formed be now removed by filtration, and an acid—say acetic acid, which does not by itself coagulate albumin—be added to the clear liquid which comes through the filter, a second bulky chocolate-coloured precipitate will be formed. Thus iodine will give with albumin in an acid state a precipitate in addition to that which it gives with albumin in an alkaline or normal state. In other words, the affinity of iodine for albumin is increased by the condition of acidity.

As bearing upon acidity in relation to the lardaceous reaction I may refer to the behaviour of indigo. A solution of sulphate of indigo loses its colour at once when alkalised by liquor potassæ; more slowly when alkalised by ammonia. If a section of healthy tissue, say of the liver, be stained with sulphate of indigo the blue colour will gradually become changed by the action of the tissue to a faint green. If a lardaceous liver be similarly dealt with, the blue staining of the lardaceous parts will remain permanent. Thus sulphate of indigo is decolorised by healthy tissue, not by lardaceous,—a difference which may with probability be attributed to the deficiency of alkali in the lardaceous. For illustrations of this difference see (4).

I must refer in particular to the results obtained by staining with methyl violet. Violet is a compound colour consisting of red and blue. For some unexplained or only partially explained reason the parts affected with the lardaceous change take and retain the red, while the blue is more feebly associated with the portions of the tissue not thus altered. Without affecting wholly to explain the decomposition of the colour by the morbid tissue, it may be worth while to note that among healthy structures some may be found which exhibit more or less of this property. By this test the distribution of the lardaceous change is more graphically and instructively displayed than by any other process I know of; and it is this upon which I shall chiefly rely in what is to follow with regard to the minute changes in the several organs.

Pathological anatomy.—I will proceed to describe in brief the changes attendant on the lardaceous condition in several of the more important organs without pretending to include all the structures and tissues in which a more persevering search might detect it.

The organ which is most extensively affected is the liver: the organ of which the affection is most productive of pathological results is the kidney, and next to this are the bowels and stomach. I will take the kidney first as the organ which is most frequently affected, and also as one which may be conveniently considered before others; because the special change in it is more easily limited and defined than where it is of greater extent.

The kidney under lardaceous disease undergoes changes, to speak first of those evident to the naked eye, which are so various that they comprise

all the results of nephritis, tubal and interstitial. The infiltration, indeed, appears to be a cause of both kinds of renal inflammation, and under it the disease takes so many shapes that it is an epitome of all. The lardaceous state is gradually produced so that the early departures are scarcely noticeable. The first is usually an exaggeration of the Malpighian bodies, which become conspicuous, and later sparkle like grains of silver sand. As the disease progresses the cortex enlarges, and many of the changes occur which are attendant upon tubal and diffuse nephritis.[1] These may even go to the form and bulk of the large white kidney, which when thus produced may be characteristically white and smooth, and have a weight, for the pair, of over 20 ounces. Sometimes the contractile process, which is due to the new fibroid tissue, overcomes the tendency to enlargement, causes the organ to shrink even below its normal size, and gives it a nodular, puckered, dimpled, or granular exterior. The elevations and depressions are apt to be of large extent and gradual slope, so as to produce an undulated rather than a nodular surface. A kidney which has thus begun to show signs of contraction may still be much enlarged nevertheless. In a case of long standing the tissue may become somewhat homogeneous and translucent, so as to suggest the comparison to wax, which has supplied one of the designations of the disease. To the naked eye the ultimate appeal lies in the action of iodine most conveniently applied as liquor iodi. This reagent brings out the Malpighian bodies as deep brown specks, and the straight vessels of the cones as lines of the same colour.

Under the microscope all the results of tubal and interstitial nephritis are apparent in their various modes and degrees. The tubes are often distended and obstructed, and new highly nucleated fibroid tissue is sometimes graphically displayed in connection with the superficial depressions. The special alteration is best seen with methyl violet; it is displayed most constantly and conspicuously in the Malpighian bodies, next upon the straight vessels of the cones, and less frequently, though often conspicuously, upon larger arteries. The lardaceous change is almost entirely vascular, and is intertubular, excepting that the Malpighian vessels may be regarded as within the tubes. The special change never, so far as I have seen, extends to the epithelium, though this is often the subject of inflammation or catarrh. The Malpighian vessel first shows the characteristic reaction, the capsule remaining to the last unaffected, save that it participates in the general fibrosis which so often accompanies the lardaceous change; the arterial knot becomes swollen and blurred, and the coils are rendered indistinct as if by some diffused exudation; it shows the reaction—whether of iodine or methyl violet—sometimes in patches, sometimes over its whole extent. The straight vessels of the cones are affected scarcely less noticeably; as sometimes also are the larger arterioles, especially those which supply the Malpighian bodies and others of similar size. The veins usually escape, though I have seen nodules or granules characteristically affected about the larger

[1] I have given a lithograph of such a kidney in my book on *Albuminuria*.

veins on the attached side of the endothelial lining. The tubes are generally unaffected, though not always; I have occasionally seen the special reaction in their walls. Though the epithelium does not participate in the special change, I have in one instance clearly recognised the iodine reaction in fibrinous casts within the tubes.[1] The interstitial tissue, apart from the blood-vessels, is usually unaffected by the special change, though I have recognised it, however rarely, in nodules or minute masses not obviously connected with vessels.

The *liver* is increased in size, nearly uniformly so; thus there is little

FIG. 13.—Abdominal swelling caused by lardaceous liver, in case of Argent referred to on p. 262.

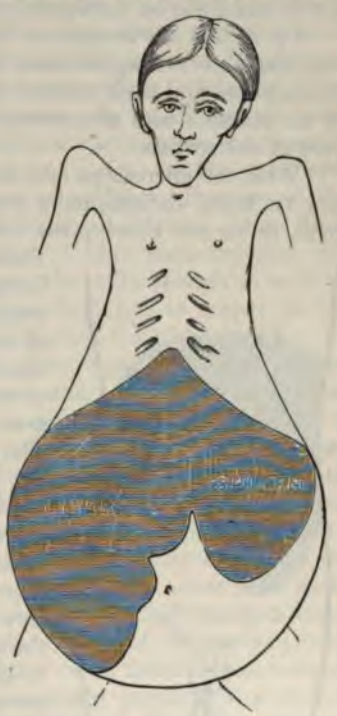


FIG. 14.—Liver as made out by touch in case of Argent.

alteration in shape. The surface usually remains smooth, and the capsule thin, and free from adhesions; unless the organ be the seat of some disease other than lardaceous, though possibly antecedent to it. The most common complication of this kind is *syphilitic;* nodules may present themselves in the liver, impinge upon the surface, and give rise to adhesions. It is possible that an abscess which has become effete after having caused lardaceous results may have left adhesions and irregularity behind. In size the liver may range from an inappreciable increase up to one such

[1] I have published a lithograph showing this in my work on *Albuminuria.*

that the lower edge of the organ will be below the umbilicus, and even below the spine of the ileum. The weight ranges up to twice or even thrice that of health. Five or six pounds are not uncommon weights for the lardaceous liver of the adult. The largest, in relation to age and stature, was that of a child (Argent) whose case I have elsewhere related (2). Her primary disease was caries of the spine. Her age was fifteen, but her stature not more than that of eight. The liver weighed 86 ounces. The right lobe descended to the fold of the groin; the left came down as low as the umbilicus, and lay in front of the spleen, from which during life it could not be distinguished.

On section the lardaceous liver, which is often also fatty, presents a very characteristic appearance, the waxy transformation of the acini being conspicuous, and their reaction with iodine remarkably extensive; so that in an extreme case the reaction, though only in dots, covers the major part of the section.

With the microscope and methyl violet the distribution of the change can be better defined, more especially when it is not extreme. In the liver, as in the kidney, the blood-vessels are the parts chiefly affected. The lardaceous tinting is often seen upon the linings of the arterioles and venules, both hepatic and portal, as if to convey a suggestion of something derived from the blood. The reaction, however, is not in all cases confined to the linings, but in the case of the hepatic arterioles sometimes permeates their coats. The middle zone of the acinus is early affected; but in advanced cases the characteristic colouring is so widely spread that it is evident that the territories, both of the hepatic and portal veins, are invaded, more particularly when these border on the region of the hepatic artery. In a very extreme case so large a proportion of the section takes the colour that it is not easy to say what structures are affected and what exempt. In such a case the small proportion of the unchanged glandular tissue remains in isolated masses, especially around the portal canals. The portal fibrous tissue is usually free from the special change which mainly affects the blood-vessels. The minute vessels in a very extreme case may be swollen and transformed almost beyond recognition, and occupy the greater part of the section. The epithelium may be fatty or atrophied, but I have never been able to make out with certainty that it gave the lardaceous reaction. It would appear that the liver gives lodgment to a larger proportion of the lardaceous matter than any other organ, and its distribution suggests that the principal channel by which it

FIG. 15.—Enlargement of liver from lardaceous disease of syphilitic origin, in case of George Hall referred to on p. 263.

is introduced is the hepatic artery. It is remarkable, considering the great increase of bulk which the liver undergoes under this disease, and the extent of the change of tissue which is evident to the naked eye, and is revealed by reagents, that the alteration in function is not greater than it appears to be—certainly it is less than in the kidney.

The *stomach* and *bowels* participate in the lardaceous condition, but they are less obviously altered than are the solid organs; and indeed less than might be expected, having regard to the pronounced symptoms which follow the extension of the disease to the alimentary canal. To the naked eye, and in an advanced case, the mucous membrane presents a certain glossy translucency, and may give the impression of being somewhat swollen; but iodine is generally needed for its sure recognition. This brings out spots and lines of a dark-brown colour, while the intermediate surface takes a yellow. It is necessary to guard against error in this respect, for the contents of the intestine may darken the reagent, while injected vessels may appear to do so. On section and with methyl violet the changes appear comparatively slight. Portions of the arterioles under the mucous coat and in the small intestine, often those belonging to the villi, take the red colour. I have seen traces of this also in the mucous structure belonging to the villi, though it may be doubted whether the faint pink colour here displayed is proper to the structure of the bowel, or due to matters absorbed from its interior.

The *spleen* may next be mentioned. This organ under lardaceous disease becomes much swollen and hardened, and acquires thick rounded edges. Having regard to its normally smaller bulk than the liver, it appears to be enlarged in a scarcely smaller ratio, though there are diseases other than lardaceous in which the spleen undergoes much greater enlargement than with this. The girl Argent already referred to, whose liver weighed 86 ounces, had a spleen which weighed 14 ounces. Having regard to the small size of the subject, it may be considered that both these organs were increased to about five times their normal weight. In the case of a man (George Hall) whose lardaceous liver weighed 6 lbs. 2 ounces, the spleen weighed 15 ounces; both the organs in this case having attained about twice their normal weight. The section of the lardaceous spleen commonly presents the special alteration most conspicuously in the Malpighian bodies; though it is not unusual to find the change, as declared by the iodine reaction, more or less diffused (3). The lardaceous condition of the spleen needs to be pointedly distinguished from the change which occurs in rickets, which is totally different; though the two have some superficial resemblance. With the aid chiefly of methyl violet the minute alterations in the lardaceous spleen may be thus described.

The lardaceous change affects the spleen in two forms, which are often intermixed. In one the Malpighian bodies are chiefly affected, and what is called the "sago spleen" is produced; in the other, the intervening tissue is affected more or less diffusely. It will suffice to describe the changes which occur in the tissue generally, and in the Malpighian bodies

in particular. The small vessels which so closely pervade the splenic tissue may almost universally take the characteristic stain; this is abundantly evident in the small arteries of about the diameter of the urinary tubules, while those of large size are sometimes affected and sometimes not. The splenic tissue between the vessels escapes, the delicately nucleated structure showing blue between the red vessels. The Malpighian bodies, when these are affected, take the colour in a striking manner, especially in their circumferential parts; the centres usually remain free. The affected ring presents an irregularly concentric linear arrangement, in which it is difficult to distinguish the proper organic structure, which looks blurred or run together, and probably consists mainly of altered capillaries. Here and there may be seen what looks like separate corpuscles affected by the special change, but this appearance may admit of doubt. It is clear that the great mass of the special change in the spleen is in the blood-vessels.

Lymphatic glands are less conspicuously affected by the lardaceous change than are many other structures; while these glands are much more altered by other pathological processes than by the lardaceous. They undergo some, but slight enlargement. Methyl violet shows that the change is chiefly in the adenoid tissue, especially about the circumferences of the masses into which it is divided. The nuclei of the lymphatic structure mostly escape, while the interstitial part is that chiefly affected. The general freedom of the cells or nuclei corresponds with what is observed in the spleen. The appearances are such as might be attributed to a general interstitial exudation. The small arteries are sometimes characteristically changed.

The *suprarenal capsules* are among the organs which are involved in the lesser degree. The changes which I have recognised in them under methyl violet have amounted to little more than a somewhat indefinite tinting of the cortical part and more distinct pink nodulation of the arterial walls.

The following statement shows the frequency with which lardaceous change was detected in the several organs and structures in 118 cases examined post-mortem at St. George's Hospital from 1877 to 1894. The examinations were made by careful observers, but without any special purpose. They cannot be regarded as exhaustive, but may be accepted as presenting not unfairly the distribution of the disease among the organs where it is commonly looked for. It is seen that the kidney is most subject to the change, then the spleen, then the liver.

Frequency with which various Organs and Structures were found to be Lardaceous in 118 cases of Lardaceous Disease examined post-mortem.

Kidney	95	Lymphatic glands	5
Spleen	76	Pancreas	1
Liver	65	Thyroid	1
Intestines	35	Œsophagus	1
Stomach	9	Testes	1
Suprarenals	9	Endocardium	1

Ætiology.—Of the immediate causes of lardaceous disease only two are clearly ascertained, though it is not impossible that others exist. The disease has often been vaguely attributed to "cachexia"; it has been associated with rickets; tubercle and cancer have been thought to be capable of causing it irrespective of any attendant suppuration; and chronic rheumatism and alcoholism have been included, though I think never very confidently, among its precursors. Rickets may be dismissed at once. This disease is attended, as I have said, with certain visceral changes which superficially resemble the lardaceous; but the conditions are essentially different, even in some respects opposite (3). I have elsewhere contrasted in some detail the rickety state with the lardaceous, and need do no more here than reiterate the conclusion that these conditions have nothing to do with each other. The two great and, possibly, only causes of the lardaceous change are suppuration and syphilis, and the greater of these is suppuration (5). In the year 1866, when analysing the antecedents of a number of cases, I was led to the belief that the loss of pus was the essential factor in the morbid process; and collateral considerations seemed to indicate that the special product was some part of the blood which was left behind after the pus had been taken from it. To emphasise this view I proposed the word *depurative* as connecting the condition with loss by suppuration; but the term, which has been charitably consigned to oblivion, is at least objectionable in this respect, that if it asserts a truth it does not assert the whole truth, for it fails to include the action of syphilis.

The only clearly recognised causes of the disease are suppuration and syphilis.

CAUSES of Lardaceous Disease in 83 Cases examined post-mortem at St. George's Hospital from 1867 to 1875. (This table was published in my book on *Albuminuria*, 2nd edition.)

	No. of cases.	Tubercle present in	Syphilis present in	Cancer present in
Continued or profuse Discharge of Pus ascertained (58 *cases*).				
Disease of bone, with discharge, caries, or necrosis	21	4	3	...
Phthisis with vomicæ	11	11	...	...
,, ,, + disease of bone	3	3	...	...
,, ,, + tubercular excavation of kidney	2	2	...	...
,, ,, + scrofulous sores	2	1	...	...
Scrofulous pneumonia with large vomicæ	1	1	...	...
Superficial scrofulous abscesses	2	1	1	...
Tubercular disease of kidney	1	1	...	...
Renal suppuration, probably calculus	1	...	...	...
Abscess of ovary opening into vagina	1	...	...	...
Gangrene or sloughing of limbs	2	..	...	...
Extensive chronic bed-sores	2	1	...	...
Pelvic abscess	1	...	..	...
Surgical operation with excessive suppuration	2	1	...	...
Ulcerating cancer of uterus, etc.	2	...	...	2
Ulcerating tumour of labium	1	..	1	...
Dysentery + syphilis	1	1	1	...
Post-pharyngeal abscess, probably syphilitic	1	...	1	...
Syphilitic ulceration of perineum	1	...	1	...
	58	27	8	2
Accumulation of Pus without Discharge (2 *cases*).				
Large collection of pus in peritoneum; general tuberculosis	1	1	...	...
Suppurating ovarian cysts	1	...	...	...
	2	1	...	...
Presumption of past Suppuration (4 *cases*).				
Old phthisis with cicatrices, but no vomicæ	3	3	...	.
Destruction of one kidney apparently by suppurative process	1	1	...	...
	4	4	...	...
Syphilis without considerable Suppuration (11 *cases*).				
Distinct syphilitic symptoms, or history	8	2	8	...
Syphilis with tubercle, or cicatrices in lung	2	2	2	...
Syphilis suspected, but not ascertained	1	1	1	...
	11	5	11	...
No evidence of either Suppuration or Syphilis (8 *cases*).				
Cause unknown	8	...	...	...
Totals, 1867 to 1875	83	37	19	2

CAUSES of Lardaceous Disease in 118 Cases examined post-mortem at St. George's Hospital from 1876 to 1894.

	No. of cases.	Tubercle present in	Syphilis present in	Cancer present in
Continued or profuse Discharge of Pus ascertained (96 *cases*).				
Phthisis with vomicæ	41	41	3	...
Phthisis with vomicæ and bone disease	2	2	...	...
Empyema discharging by surface or bronchi	3	...	...	...
Disease of spine discharging superficially	12	5	...	...
,, hip-joint or pelvis ,,	18	6	...	...
,, skull, knee, femur, tibia ,,	6	...	1	...
Scrofulous disease of kidney causing lumbar abscess and empyema	1	...	...	...
Scrofulous abscesses of testicles, excision of both	1	1	...	...
Extensive bed-sores of long standing	1	...	...	...
Tropical dysentery with much ulceration	1	...	...	...
Dysentery + abscess of liver discharging superficially	2	...	1	...
Dysentery + abscess of liver and empyema (pus shut up)	1	...	...	...
Hydatid abscess of liver opened superficially	1	...	...	...
Albuminuric ulcer of bowel, abscess in peritoneum and profuse discharge by bowel	1	...	...	...
Fæcal abscess discharging superficially	1	...	...	...
Pelvic abscess discharging superficially	1	...	...	...
Calculous pyelitis. Nephrotomy	1	...	...	...
Stones in kidneys and bladder. Cystitis. Lithotomy	1	...	...	...
Long-standing cystitis; final sloughing of bladder	1	1	...	...
	96	56	5	...
Accumulation of Pus without Escape (1 *case*).				
Empyema in child of 2½ years, not discovered in life	1	...	...	...
	1	...	...	...
Presumption or Evidence of past Suppuration (6 *cases*).				
Scrofulous destruction of one kidney	2	1	...	...
Sinus opening into rectum leading from old mass of cicatricial tissue in place of ovary	1	...	...	...
Scrofulous abscesses about neck thirty-one years before death	1	1	...	...
Destruction of one kidney by old disease of uncertain character	2	...	1	...
	6	2	1	...
Syphilis without considerable Suppuration (12 *cases*).				
Syphilis with gummata or gummatous cicatrices	8	1	8	...
History of syphilis, but no gummata	3	1	3	...
Syphilis suspected, but not ascertained	1	...	1	...
	12	2	12	...
No Evidence of either Suppuration or Syphilis.				
Cause not ascertained	3	...	...	...
Totals, 1876 to 1894	118	60	18	...

SUMMARY of the two preceding Tables, showing the Causes of Lardaceous Disease in 201 cases taken from the Records of St. George's Hospital from the year 1867 to the year 1894.

	No. of cases.	Tubercle present in	Syphilis present in	Cancer present in
Continued or profuse Discharge of Pus ascertained (154 *cases*).				
Phthisis with vomicæ, including excavating pneumonia	53	53	3	...
,, ,, ,, + bone disease	5	5	...	...
,, ,, ,, + tubercular excavation of kidney	2	2	...	...
,, ,, ,, + scrofulous sores	2	1	...	...
Disease of bone with consequent discharge	57	15	4	...
Scrofulous disease of kidney	2	1	...	...
,, ,, testicle	1	1	...	...
Superficial scrofulous abscesses	2	1	1	...
Discharging empyema	3	...	...	...
Pelvic or ovarian abscess	3	...	...	...
Pyelitis or cystitis, with or without stone	4	1	...	...
Dysentery	2	1	1	...
,, + abscess of liver	3	...	1	...
Hydatid abscess of liver	1	...	...	...
Peritoneal abscess from albuminuric ulcerations of bowel	1	...	...	...
Fæcal abscess	1	...	...	...
Cancer of uterus	2	...	...	2
Syphilitic ulceration or abscess	3	...	3	...
Gangrene or sloughing of limbs	2	...	...	.
Surgical operations	2	1	...	..
Bed-sores	3	1	...	...
	154	83	13	2
Accumulation of Pus without Exit (3 *cases*).				
Empyema	1	...	...	...
Pus in peritoneum	1	1	...	...
Suppurating ovarian cysts	1	...	...	...
	3	1	...	...
Presumption of past Suppuration (10 *cases*).				
Old phthisis with cicatrices, but no vomicæ	3	3	...	...
Destruction of one kidney presumably by suppuration	5	2	1	...
Sinus opening into rectum	1	...	...	...
Scrofulous abscesses about neck thirty-one years before death	1	1	...	...
	10	6	1	..
Syphilis without considerable Suppuration (23 *cases*).				
Distinct evidence of syphilis	19	4	19	...
Syphilis with tubercule or tubercular cicatrices in lungs	2	2	2	...
Syphilis suspected but not ascertained	2	1	2	...
	23	7	23	...
No evidence of either suppuration or syphilis (cause unknown)	11	...	...	...
	201	97	37	2

SUMMARY of preceding Table, showing causes of Lardaceous Disease in 201 cases.

	No. of Cases.
Discharge of Pus	154
Accumulation of pus without discharge	3
Presumption of past suppuration	10
Syphilis without considerable suppuration	23
Cause unknown (no evidence of suppuration or syphilis)	11
	201

The present sketch of the etiology of the disease is based on a collection of 201 cases obtained from the post-mortem and case-books of St. George's Hospital between the years 1867 and 1894. The cases are in two sets, the first of which, from 1867 to 1875, was published in the second edition of my book on *Albuminuria;* the second series, from 1876 to 1894, was compiled for the purpose of this article: it is believed that the two together give a fair account of the genesis of the disease as it occurs in a general hospital. It is to be noticed that, being based on post-mortem observation, many of the lesser degrees of the change are included which could not have been discovered clinically. During the comparatively recent period covered by the cases adduced considerable attention, I think I may say especially at St. George's, has been directed to the morbid change in question; and I believe that the records concerning it are fairly trustworthy. To proceed to the examination of the tables: first, as regards suppuration it will be seen that this process, with or without escape, recent and obvious, or ancient and presumed, is presented in 167 of the 201 cases; that is, in over three-fifths. Pulmonary phthisis and disease of bone are both extremely frequent, and about equally so in this relation; each accounts for over a quarter of the total. Syphilis presents itself as the ostensible cause in 23 of the 201, or in about one-ninth. If we take all the cases in which syphilis was recognised as in any way mixed up with the case, though possibly not its main feature, we must give it a place in 37 of the 201, or about a fifth. It is not easy to estimate exactly the influence of syphilis in this matter; it is obvious that a person may have had this disease and may die of another entirely unconnected with it; on the other hand, it is certain that this change, though possibly an important factor, must be often overlooked. Tubercle and cancer do not appear as lardaceous antecedents excepting with suppuration as an intermediary. Cancer may be briefly dismissed; it occurred but in two instances. Tubercle, on the other hand, was exceedingly frequent; it was present in nearly half of the whole number. Associated as tubercle is with chronic suppurative processes this could hardly be otherwise; but, independently of purulent formation, there is no evidence that it causes lardaceous disease. There is abundant evidence, on the other hand, that suppuration without tubercle or any recognised constitutional taint is effective in this respect; witness the considerable

number of instances in which the organic change has ensued upon non-tuberculous empyema, abscesses originating within the abdomen, abscesses of the liver, dysentery, and extensive bed-sores in which there has been no complication of the kind referred to.

Among the problematical causes of the lardaceous change is malaria. Malaria is common in India, so is the lardaceous disease, if we may judge by the frequency with which it is displayed by returned Indians. Malarial fevers cause wasting and cachexia, and have a special effect in destroying the corpuscles of the blood. Lardaceous disease would seem to be a possible result, but I can find no evidence that it is an actual one. Malaria appears to exert its destructive influence mainly upon the red corpuscles; lardaceous disease when produced by suppuration must be due to the loss of the white. I have had especial regard to ague and remittent fever in examining the records to which I have so often referred, and out of the 201 cases I can find but three where there was a distinct malarious history; in one of these there was a history also of dysentery, in another of dysentery together with abscess of the liver; so that only in one instance is malaria presented as the ostensible cause, and even in this we have no assurance that it was the actual one.

While upon the topic of malaria a question presents itself: Does the influence of a tropical climate, irrespective of any special disease, produce or promote lardaceous change? I think there can be no doubt that the influence of the tropics acts as a predisposing or co-operating cause, though not in itself sufficient; suppuration is apt to be profuse in hot climates; there are many facilities for the acquirement of syphilis, and the imperfect nutrition of the European must lessen his power of resistance where nutrition is concerned.

Another question arises on the etiology of lardaceous disease: Does this condition ever ensue as the consequence of protracted albuminuria apart from suppuration or syphilis? Lardaceous kidneys take almost every shape which is covered by the name of Bright. A chronic granular kidney, which looks at first sight as if it might be gouty, or a large white kidney which without testing might pass as the result of simple nephritis, may give the lardaceous reaction. Is the pathological change which this indicates a mere superaddition to disease of another kind, or is it always to be regarded as the primary or at least an independent condition? in short, is long-continued albuminuria to be reckoned among the causes of the lardaceous change? I think this question must be answered in the negative. The lardaceous infiltration is capable of causing renal inflammation, chronic and acute, tubal and interstitial; and it may be at the bottom of almost any type of albuminuric disease. The history of such cases as appear mixed will almost always display some connection with suppuration or syphilis; while, on the other hand, even the most protracted cases of renal disease which distinctly originate in other causes fail to acquire any lardaceous complication.

Suppuration, to give rise to lardaceous consequences, must as a rule be long continued; hence it comes to pass that so many cases of this

origin are connected with tubercle and disease of bone. In looking through a large number of cases, I find that the suppurative process was dated back from death for periods ranging in the majority from one year to ten: though these limits in a few were fallen short of, or exceeded. As an instance in which comparatively brief suppuration was thus effective, I may mention one in which I satisfied myself that the Malpighian bodies of the kidney gave the reaction after only three weeks of suppuration connected with amputation of the thigh. The discharge was most profuse. The subject was a previously healthy young man, whose limb was removed immediately after a compound fracture. Death was due to pyæmia (6). The lardaceous change evidently comes on during the presence of the suppuration; though it may not be discovered until it has continued for many years, or even until many years after it has ceased. A patient who died of renal disease of this nature had had a discharging sinus for twenty-four years; another, whose death was mainly due to phthisis, but had also lardaceous symptoms, albuminuria, and diarrhœa, had a history of phthisis extending over twenty years. In a case in which the renal results of the disease proved fatal at the age of 58, nothing more recent was found to account for it than suppurating cervical glands at the age of 27, and ancient lung disease which had been attended with habitual winter cough; it was represented at death only by caseous deposits.

In regard to this effect of suppuration, it is striking how individuals differ in their power of resisting it; wasting and exhausting processes tell so differently upon different persons, that what is harmless to one may be fatal to another. It is obvious that suppuration occurring together with acute illness or general disturbance of health is more mischievously effective than where the constitutional state is less involved; and one man is more resistive than another, according to his strength of constitution, the goodness of his stomach, or the advantages of his surroundings. When the condition arises from syphilis, which it may do quite independently of any suppurative process, the organic change is a late, or what I suppose would be called a tertiary result. Among the cases (118 in number) which I have abstracted from the St. George's records for the purpose of this paper, I find that the time of the infection before death, so far as it could be ascertained, ranged from three years to thirty-two years; and it is possible that a larger experience might increase the range in both directions. A patient in whom the syphilis was apparently congenital died of lardaceous disease at the age of 21.

Subjects of Lardaceous Disease.—The subjects of the disease as presented in hospital practice are more often male than female, in the proportion shown below. It is not to be supposed that the disease has any sexual preference, but many of the causes to which it has been traced affect the male more than the female. Men are more amenable to violence and exposure than women, and are more often syphilitic.

With regard to age the disease is rare under 10 or after 50; it is commoner between 20 and 40. It may be said that its chosen march is the prime of life. As the slow result of antecedent mischief it seldom

presents itself very early in life; while, since the diseases which it follows mostly belong to the early and middle periods, advanced age is usually exempt.

Sex of 118 Fatal Cases of Lardaceous Disease examined post-mortem at St. George's Hospital.

Males, 87. Females, 31.

Age at Death of 116 Cases of Lardaceous Disease examined post-mortem at St. George's Hospital.

Age at Death. Years.	Number of Cases.
0 to 10	4
11 ,, 20	20
21 ,, 30	31
31 ,, 40	36
41 ,, 50	17
51 ,, 60	8
61 ,, 70	0
Over 70	0
	116

Symptoms.—As the lardaceous condition is, in the first place, general to the blood, and the tissue localisations secondary or consequent, so there are constitutional manifestations of the disease irrespective of the several organic lesions. Syphilis has frequently left its mark. Together with this, or apart from it, the effects of the tropics are often to be discerned in the leanness and brownness which testify alike to both influences. The wasting results of various chronic and exhausting diseases are apparent, and sharp features and spindle shanks are often conjoined with a prominent abdomen. The *pallor luteus* is more common than the ivory pallor of recent and simple renal disease, though this form of white swelling is sometimes to be noticed when the lardaceous change is purely renal, is recent, and of suppurative origin. The condition is often to be recognised at a glance: the crooked back or obvious joint disease, the look of travail or of sad experience, supply a ready explanation to the albuminuria, the dropsy, or the visceral enlargements which are perhaps the ostensible evils which have brought the patient and the physician into contact. It rarely happens that the diagnosis is a matter of doubt; apart from the antecedents, diarrhœa, polyuria, and thirst are indicative, as also is the distribution of the œdema, which is more like that of heart disease than of kidney disease in the respect that it affects the ankles more than the face.

The symptoms of lardaceous disease as proper to the separate organs will be discussed in connection with the diseases of each; here it is only necessary to provide a somewhat general description with but brief reference to the organs in particular.

The outset of the disease is usually gradual and even imperceptible. The most prominent consequences are albuminuria, diuresis, thirst, diarrhœa, vomiting, œdema or wasting according to circumstances, or possibly both together; enlargement of the liver and spleen, and prostration. In the majority of cases the renal changes are the earliest, as they are the most constant. An analysis of 118 cases from the post-mortem records of St. George's, which were collected without special regard to any particular organ but included all in which the lardaceous change was found after death in any situation, presented 32 in which, though the special change was apparent after death, no symptoms of it were detected during life. Of the 86 cases in which symptoms had been noted which were attributable to the special change, these symptoms were referrible to the kidneys in 76. In 67 of these the urine was recorded as albuminous; in 57 œdema was present. Though there were several cases, some of them in surgical wards, in which renal symptoms, mostly œdema, were present, but in which the state of the urine was not observed or recorded, there is but one instance among them in which the urine was definitely stated as not containing albumin; this was a case of phthisis, and though the kidneys showed lardaceous change, it is possible that the œdema may not have been truly renal. In very early renal changes of this nature the urine may be not yet albuminous; an early state of the transformation may be discoverable after death where no symptoms had as yet reached the surface.

Next in frequency to the renal symptoms come those which point to the intestinal complication. Diarrhœa was recorded in 52 of the 118 cases of which I have written, and was largely concerned in causing death in 21. Of all the symptoms of lardaceous disease diarrhœa has the greatest diagnostic significance and the greatest vital importance. It is not possible quite to separate the diarrhœa due to a lardaceous state of the bowel from a condition with which it is often associated, namely, tubercular ulceration. Vomiting is a less frequent accompaniment of lardaceous disease than diarrhœa; it is generally due to the special change in the stomach itself, sometimes it belongs to the uræmia which pertains to the renal complication. In the series of cases I have referred to, vomiting was noted in 28 instances; in 5 of these it was probably uræmic.

In the lardaceous process the liver and spleen are apt to become enlarged, seldom extremely, but enough to be evident to the hand, and to come within the clinical purview of the disease. At page 261 is an outline showing what may be considered the maximum of hepatic swelling from this cause; other figures are annexed which display such moderate visceral enlargements as are more commonly met with. Tangible or at least great enlargement of either of these organs is present only in a minority of cases—that of the spleen less often than that of the liver; and it may be added that though tangible organic enlargement is by no means common, yet symptoms in connection with the hepatic or splenic changes are more infrequent still. It is, indeed,

a matter of surprise, when we see the extent and frequency of the alterations in the liver and spleen (more particularly of the liver) as displayed in the post-mortem room, that corresponding symptoms are not more conspicuous. It is not difficult (as will be elsewhere shown) to gather from a wide experience many instances of hepatic enlargement with some of which there have been disturbances of function; but as compared with renal or bowel symptoms those relating to the liver are in a very small minority. Jaundice and leucocythæmia, as the results of lardaceous disease, are both extremely uncommon. In the series of 118 cases, putting aside mere enlargement, there were but six in which symptoms occurred which could be attributed to the liver. In one case, complicated with hydatid abscess of the liver, which was also lardaceous, there was jaundice; there was none in any other case. In all the six there was ascites; but in four of them it is possible that this symptom may have been partly renal; for in them there was œdema, apparently of this nature, together with albuminuria. With regard to the spleen, the enlargement, if present, is not extreme, nor does it approach what is commonly found with leucocythæmia: the symptoms of the splenic participation are not marked beyond the general consideration that this, together with the hepatic change, is concerned in bringing about the general look and state which is expressed by a word which is the more useful because not too exact—cachexia.

Death is more often directly due to the antecedent disease than the lardaceous consequence. Taking cases as they present them[illegible] the post-mortem room, it appears that in a considerable propo[illegible] them no lardaceous symptoms had as yet declared themselves [illegible] evidences of the change were apparent after death. It [illegible] that the morbid process must reach a certain point before [illegible] noticeably with function. The following table indicates [illegible] cause of death in 118 cases where the lardaceous [illegible] covered afterwards. It is seen that the lardaceous [illegible] chief cause of death in about a third of the num[illegible] the rest it was brought about directly by the [illegible] phthisis, and disease of bone being the most num[illegible] understood that as lardaceous disease was pre[illegible] must have contributed to the fatal issue in [illegible] as the main or chief cause. In one or [illegible] obviously as to require special mention [illegible] order; though the primary disorder [illegible]

etive are those
ght by the sea-

DICKINSON.

87. New Sydenham
KINSON, W. H. *Med.-*
867, vol. l. p. 39, plate
Albuminuria, 2nd edit.
p. 3.—8. FAGGE, C.
of Liver, vol. ii. p. 167.
KEKULÉ. *Virch. Arch.*
ign Medico-Chirurgical
—13. HAMILTON, D. J.
ch. Archiv, Bd. lxv. S.
gy, p. 65. Translated by
h. u. path. Anat. May 20,
—18. LEA. *Chemical Basis*
f Committee on Lardaceous
KEL, H. *Charité-Annalen*,
1884, vol. x. series iii. p. 315.
gy, vol. i. p. 43. New Syden-
st. 1846, i. S. 445.—24. RUD-
8.—25. VIRCHOW. *Virchow's*
Cellular Pathology. Translated
Heilk. ii. S. 481.—28. WEBER,
1895.—29. WICHMANN, G. *Beitr.*
d, with extensive bibliography.—
es iii. vol. ii. p. 105; 1865, series iii.
ological Anatomy, vol. i. p. 90, trans-

W. H. D.

place, that the lardaceous deposit may be removed spontaneously, or the condition rectified by the efforts of nature. A tendency to recovery is to be recognised, though not much reckoned on. After death organs are occasionally found to display all or nearly all the lardaceous characters except the reaction which appears to have been abolished as the disease receded. A marked history of renal disease of lardaceous origin may eventuate in a fibrotic kidney which no longer presents the special reaction; the kidney in this case has been permanently damaged, though no longer lardaceous: there is reason to believe that this change may present itself to a slight but decided extent and pass away, leaving neither functional disturbance nor visible change. I have often thought that temporary suppuration may produce temporary lardacity. These considerations encourage surgical endeavours to abolish suppurative lesions. Lardaceous patients, unless the disease be extreme, retain a considerable amount of healing capacity, and often do well after operations. It is certain that lardaceous conditions are now less frequent and less extreme than they used to be twenty years ago; a change which may be attributed to the increased use of iodide of potassium and the introduction of antiseptic surgery. Iodide of potassium is not only of obvious use in controlling the later effects of syphilis and preventing the lardaceous and other complications, but it exerts a distinctly curative action upon lardaceous disease which has been left behind or has ensued upon a remote specific infection. When renal or other visceral disease of this nature can be traced even remotely to syphilis, experience gives us encouragement to urge the remedy in considerable doses and for a long time; chronic and apparently hopeless renal disease sometimes yields to such perseverance. The treatment must extend over years, beginning with substantial doses—say 20 or 30 grains a day, reducing as time goes on or the patient becomes intolerant. I often give iodide of iron with the potash salt. I have seen the best results in renal cases from the combined influence of iodide of potassium and a southern winter resort—the Riviera or Egypt. In such a climate the iron may be dispensed with.

Supposing there be no question of syphilis or of surgery, the lardaceous state must be generally treated with restoratives, which may be the more liberally administered the less renal the stress of the disease. Should uræmia threaten, it is not necessary to insist that it may be hastened by over-dieting. But, as a rule, the diet may be liberal, and all the means employed in favour of the general health, on general principles, which are within reach. Sea-air and country air are beneficial, especially sea-air—of the "bracing" kinds of which Margate may be taken as an example. Medicines of the tonic class are beneficial—iron, perhaps best as iodide, quinine, and cod-liver oil; especially if the case be tubercular.

Looking at the solubility of the morbid deposit in alkalies, and at the deficiency of potash in its composition, I formerly gave potash with a remedial aim. If so given it should be as liquor potassæ upon an empty stomach; but the good is less apparent than is that of the drugs before-

mentioned, and, perhaps, of all remedies the most effective are those which come from the laboratory of Nature, and are brought by the sea-breezes.

W. HOWSHIP DICKINSON.

REFERENCES

1. COHNHEIM'S *Lectures on General Pathology*, vol. ii. p. 687. New Sydenham Society.—2. CORNIL. *Archives de physiol.* 1875, p. 671.—3. DICKINSON, W. H. *Med.-Chir. Trans.* vol. liii. p. 370.—4. *Idem. Med.-Chir. Trans.* 1867, vol. l. p. 39, plate 2.—5. *Idem. Path. Soc. Trans.* 1879, vol. xxx. p. 511.—6. *Idem. Albuminuria*, 2nd edit. pp. 234-275; 1877.—7. DUPRÉ. *Path. Soc. Trans.* vol. xxxii. p. 3.—8. FAGGE, C. HILTON. *Path. Trans.* 1876, p. 324.—9. FRERICHS. *Diseases of Liver*, vol. ii. p. 167. Translated by New Sydenham Society.—10. FRIEDRICH and KEKULÉ. *Virch. Arch.* Bd. xvi. S. 50.—11. GRAINGER STEWART. *British and Foreign Medico-Chirurgical Review*, 1866.—12. *Idem. Bright's Disease*, 2nd edition, 1871.—13. HAMILTON, D. J. *Text-Book of Pathology*, vol. i. p. 167.—14. JURGENS. *Virch. Archiv*, Bd. lxv. S. 189.—15. KAHLDEN, VON. *Methods of Pathological Histology*, p. 65. Translated by H. M. Fletcher.—16. KRAWKOW. *Centralblatt f. allg. Path. u. path. Anat.* May 20, 1895.—17. KYBER. *Virchow's Archiv*, Bd. lxxxi. S. 270, 420.—18. LEA. *Chemical Basis of the Animal Body*, p. 47.—19. MARCET, W. "Report of Committee on Lardaceous Disease," *Path. Trans.* 1871, vol. xxii. p. 1.—20. MECKEL, H. *Charité-Annalen*, 1853, iv. 2.—21. PAVY, F. W. *Guy's Hospital Reports*, 1884, vol. x. series iii. p. 315.—22. RINDFLEISCH. *A Manual of Pathological Histology*, vol. i. p. 43. New Sydenham Society.—23. ROKITANSKY. *Lehrb. d. path. Anat.* 1846, i. S. 445.—24. RUDNEFF and KÜHNE. *Virch. Arch.* Bd. xxxiii. S. 66.—25. VIRCHOW. *Virchow's Archiv*, Bd. vi. S. 268, 416; viii. S. 364.- 26. *Idem. Cellular Pathology.* Translated by Dr. Chance, p. 367.—27. WAGNER, E. *Arch. d. Heilk.* ii. S. 481.—28. WEBER, F. P. *American Journal of Medical Sciences*, Nov. 1895.—29. WICHMANN, G. *Beitr. path. Anat. v. allg. Path.* 1893, Bd. xiii. S. 487, with extensive bibliography.—30. WILKS, S. *Guy's Hospital Reports*, 1856, series iii. vol. ii. p. 105; 1865, series iii. vol. ii. p. 65.—31. ZEIGLER'S *Text-Book of Pathological Anatomy*, vol. i. p. 90, translated by MacAlister.

W. H. D.

DISEASES OF ALIMENTATION AND EXCRETION

GENERAL PATHOLOGY OF DIGESTION

THE process of digestion is in part mechanical and in part chemical. The first part of the process is required to reduce the less soluble portions of the food to a state in which the digestive secretions can act upon them. Moreover, it ensures the due propulsion of the alimentary mass, and the submission of it to the several agents which affect it in turn as it traverses the whole length of the digestive system. The chemical changes are effected by the action of various digestive secretions, poured out by special glands at different parts of the alimentary canal. These secretions subject the constituents of the food, chiefly the hydrocarbons and proteids, to changes which convert them into bodies easy of absorption and fit for assimilation. So interdependent are the various parts of the process that failure either of the mechanical or of the chemical factors leads sooner or later to disturbance of the whole. It is therefore convenient to consider separately the several portions of the digestive tract in which the chief alterations of the food-stuffs occur; yet, when use is made of such expressions as "oral," "gastric," or "intestinal digestion," it must be remembered that a disturbance of any one of these three processes will in more or less measure disturb the integrity of the rest.

I. ORAL DIGESTION.—In the mouth the coarser portions of the food are broken up and subjected to a process of trituration, whilst the softer masses become more thoroughly disintegrated, and are incorporated with the saliva by means of the teeth, the tongue, and the buccal muscles.

The Saliva, as it appears in the mouth, is not the pure secretion derived from the three pairs of true salivary glands—the parotid, the submaxillary, and lingual, but is largely mixed with mucus from the glands of the buccal mucous membrane, with portions of food in a state of decomposition, and with numerous micro-organisms. When, however, as in the physiological laboratory, saliva is obtained pure, it appears as a frothy, viscid, and transparent fluid of a specific gravity ranging from 1·002 to 1·006. Its normal reaction is alkaline, but sometimes owing to decomposition it is acid. Under the microscope certain so-called "salivary corpuscles" are seen. These chiefly consist of altered leucocytes and squamous epithelium from the oral mucous membrane. Large numbers of micro-organisms are also visible. The average composition of saliva may be stated as yielding in 1000 parts: water, 994·4;

solids, 5·6. The solids consist of about 3·5 of soluble organic matter and epithelium and 1·6 of salts. The soluble organic matter comprises a proteid resembling serum albumin, mucin, globulin, a diastatic enzyme (*ptyalin*), traces of a proteolytic ferment probably pepsin, and certain extractive matters, such as urea and leucine. Lactic acid is occasionally found but is indicative of disease. The salts consist of the alkaline and earthy phosphates, carbonates and chlorides; sulphocyanate of potassium is an occasional ingredient of human saliva. Free ammonia can nearly always be found in oral saliva, being probably the result of decomposition. It is said that nitrates are also to be found. The three pairs of salivary glands—the parotid, submaxillary, and lingual—are alone concerned in furnishing the diastatic enzyme of salivary digestion. A mixture of the secretions of all three pairs of glands is more active than the secretion of any one pair taken alone, but the secretions of these pairs differ one from another. The saliva obtained from the parotid is rich in diastatic enzyme, but contains only a trace of mucin; the submaxillary secretion is rich in mucin, and contains but traces of the diastatic ferment. The sublingual saliva in the human subject has not been investigated; but in dogs it has been obtained in a viscous mass resembling frogs' spawn. The physiological effects of the stimulation of the nerves supplying these two glands have been thoroughly investigated and are recorded in the physiological text-books. Here it is only necessary to summarise the results. Stimulation of the parotid by Jacobson's nerve produces a watery secretion containing little proteid matter, diastatic ferment or salts. Irritation of the sympathetic causes no secretion; but irritation of Jacobson's nerve and the sympathetic at the same time gives rise to an abundant supply in which the organic constituents abound. The salts, however, are but slightly increased. Stimulation of the chorda tympani produces an increased flow of saliva from the submaxillary gland; if, however, the stimulation be directed exclusively to the sympathetic filament, the secretion, though abundant, becomes thicker and more gelatinous than that which flows on stimulation of both filaments. Section of the nerves without stimulation often leads to the discharge of a thin aqueous secretion for days and weeks together—the so-called "paralytic saliva."

(*a*) *Alteration of reaction.*—The normal alkaline reaction of the saliva has an undoubted effect on the gastric secretion. Claude Bernard found by experiment that it is the best stimulant to excite the flow of gastric juice. On the other hand, the anorexia that attends all cases of salivation may be due to the dilution and neutralisation of the gastric juice by large quantities of alkaline saliva. The reaction of the saliva is sometimes acid, even in persons of apparent health. This is probably due to acid fermentation occurring in the mouth. It has repeatedly been stated that in diabetes the reaction is acid, and that this is due to the presence of lactic acid. Dr. Gamgee from observation of two confirmed cases, in both of which he found a marked alkaline reaction, has denied this statement. One of us (Ralfe) has noticed an acid reaction on several

occasions, but the phenomenon is not constant: when it occurs it is probably due to acid fermentation, and not to the secretion of acid saliva.

(*b*) *Alterations in quantity* are apparent in the dripping, moist, dry, or parched mouth, according as the secretion is profuse or scanty. The average daily quantity is about 1500 grains. Under normal circumstances an increased flow of saliva follows reflex stimulation by the sight and smell of food, but there may also be more direct causes. A form of salivation, which in its exaggeration resembles "the watering of the chaps" of a healthy appetite, is the profuse watery discharge which, with a similar discharge from the gastric glands, constitutes "water brash." In certain lesions of the nervous system the secretion is at times thin, watery, and paralytic, or thick and viscous—changes which may be demonstrated experimentally by the section of nerves. Salivation often occurs in hysterical and in pregnant women. It also occurs in diseases and injuries to the mouth, such as thrush, scorbutic, tuberculous, syphilitic, malignant ulcers; or from corrosive poisons. Certain drugs (mercury, iodine, jaborandi), acting through the nervous system, have like effects. In insanity, again, and in teething children salivation is no unusual symptom. Cases of apparent salivation may be due to difficulty of swallowing, of which an instance may be cited in cases of bulbar paralysis; although the quantity and the long continuance of the flow in this disease prevent us from regarding it as equivalent to the so-called "paralytic secretion." The evils of long-continued salivation have been attributed to (α) a diminution of the amount of diastatic enzyme, which certainly has a low percentage ratio in this condition; (β) the drain of water from the body; (γ) the dilution and neutralisation of the gastric juice by the constant swallowing of large quantities of alkaline saliva. These are perhaps the most important factors at work in causing the weakness and emaciation of long-continued salivation.

Diminution of the salivary secretion, as Cohnheim suggests, can only occur as a subordinate pathological incident. He points out that to stop all secretion either the destruction of all the glands or the destruction of all nervous influence upon them would be necessary. The only interpreting symptoms of which we have any positive knowledge is that pyrexia checks the parotid secretion. But even then the power of converting starch into sugar is never quite absent, and the diminution of the secretion is probably in respect only of its watery constituent. A similar diminution of the secretion is seen in the thirst and dryness of mouth that attend any profuse drain of water from the system, as for instance in diabetes. In this case the thirst is not caused by the presence of sugar in the circulation, but by the diuresis; for if we control the diuresis by opiates, the thirst and dryness of the mouth and the urinary excretion fall together, though the sugar excretion may still continue excessive.

(*c*) *Activity of the aiastatic enzyme, commonly called ptyalin.*—In newborn children this ferment appears only in the parotid; it is not developed in the submaxillary gland or pancreas till after two months of age. Recent

investigations have shown that the sugar which results from the action of saliva on starch is maltose; the reaction may be expressed by the following formula:—

$$\underset{\text{Starch}}{10\ C_{12}H_{20}O_{10}} + 8\ H_2O = \underset{\text{Maltose}}{8\ C_{12}H_{22}O_{11}} + \underset{\text{Dextrin}}{2\ C_{12}H_{20}O_{10}}$$

Whether any sugar other than maltose is formed by the action of saliva on starch is not yet known. Dr. Sheridan Lea in his experiments did not obtain any positive evidence that such is the case. The diastatic action of saliva is proved by the disappearance of the starch and its conversion into sugar.

Alterations in amount and activity of ptyalin.—As we are unable to obtain the ferment in an absolutely pure state, so as to determine it by weight, we have to ascertain its "diastatic value" by means of Sir William Roberts' method of "diastasimetry." This method depends upon the final disappearance of all dextrins (which are coloured yellow by iodine), the solution containing only maltose and achroo-dextrin, which are not so affected. This method of estimating the activity of a diastatic solution on a given standardised starch solution is probably the most accurate which we possess. The salivary ferment acts best in neutral solutions, but it is efficient also in alkaline and even in slightly acid media, when the degree of acidity does not exceed 0·023 per cent of mineral acid and 3 per cent of organic. Stronger solutions than these put a stop to its action. The ferment, therefore, retains its power of converting starch in the stomach so long as the gastric juice does not rise beyond a certain point of acidity. Even if the degree of acidity be so high as to arrest it, its activity may be restored by careful neutralisation. The action of the saliva on the starches varies with the amount of cellulose they contain, and also with their state of solubility; as, for instance, whether boiled or raw. Certain other substances hinder its action: tea, by virtue of the tannin it contains, has a strong inhibitive effect upon it; but the retarding influence of coffee and cocoa is but slight. Alcohol, if taken in large quantities, puts a stop to starch conversion. Sir William Roberts has pointed out a rational way of dealing with the inhibitory effects of tea and alcohol by suggesting we should eat first and drink afterwards. There are certain drugs, also, which arrest and soon destroy the action of the salivary ferment: common salt, ammonium chloride, and some other substances, on the other hand, increase its activity. These points will be considered further on when the action of drugs on the salivary secretion is discussed more fully. The fermentative influence of saliva upon starch in the mouth is so very brief that we shall defer a consideration of the results of the malassimilation of this substance until we treat of its further digestion in the stomach, and of the action of the pancreatic ferment in the intestine.

(*d*) *Mucin* is secreted in abundance by the submaxillary and sublingual glands; it gives to saliva its viscidity, and consequently aids in the act

of deglutition. The secretion of mucus is increased when the mouth is inflamed, and in cases of mumps. Such catarrhal mucus contains an excess of albumin, lymph corpuscles, and epithelial débris. The same variation is observed in the earlier stages of ptyalism. An increase of mucus, however, as Cohnheim points out, is not so important a factor in ill health as the opposite condition of diminution, since buccal digestion is not interfered with. A dry mouth, however, has always been considered a sign of serious illness. Fever patients, Cohnheim adds, can keep their mouths moist so long as they are freely supplied with water; a loss of the sense of thirst or inability to satisfy it is ominous of approaching coma. The mucin of the saliva has the further property of forming the peculiar stringy masses hawked up from the stomach in some forms of dyspepsia, chiefly the alcoholic; but this regurgitation may occur whenever acetic acid fermentation is set up by the ingestion of substances prone to give rise to it. From its occurrence in the early morning, after first rising, Frerichs was led to suppose that it is due to the action of free acetic acid upon the mucin of the saliva swallowed during the night, converting it into stringy and ropy masses. It speedily subsides on the administration of remedies which stop the fermentative changes in the stomach.

(*e*) *Salts.*—These chiefly consist of alkaline chlorides, mixed with earthy phosphates and occasionally also with carbonate of lime. Sulphocyanate of potassium is a variable constituent of the saliva. Dr. Fenwick has made some important observations which point to this salt being a product of the secretion of the salivary glands, and has found that it is much increased in quantity in gout and rheumatism, but deficient in chorea, acute atrophy of the liver, and inanition. Other observers believe that the salt arises from decomposition in the oral and buccal cavities. The characteristic test for the presence of sulphocyanate of potassium is the production of a blood-red colour by the addition of perchloride of iron, which colour is immediately destroyed by a solution of perchloride of mercury.

(*f*) *Action of drugs on salivary secretion.*—The salivary secretion can be increased, diminished, or altered in chemical composition by the administration of certain drugs: (*a*) An increase in the amount of saliva may be brought about, either in a reflex manner by means of such substances as ether, chloroform, acids, alkalies, mustard, pellitory, ginger, and horse-radish, which stimulate the mucous membrane of the mouth or stomach, or by the direct stimulation of the secretory nerves or of the glandular structures by such drugs as pilocarpine, nicotine, muscarine, physostigmine, mercury, and iodine; (β) in like manner the reflex excitability of the nervous centre controlling the secretion can be diminished by opium, morphine, codeine, and bromides; or the nerve-endings in the glandular tissue may be paralysed by atropine; (γ) lastly, certain drugs are habitually excreted by the salivary glands; of these the most important are the iodides, which can be detected in the saliva within fifteen minutes of their entrance into the stomach. It is also

noteworthy that both iron and quinine, when combined with iodine, are eliminated in the saliva. Pilocarpine renders the secretion more liquid, but at the same time diminishes its diastatic action.

(*g*) *Influence of disease on the salivary secretion.*—In addition to the alterations in quantity and quality which the salivary secretion is prone to exhibit in various diseases, it is also found that the amount of the sulphocyanide of potassium contained in it varies considerably in different disorders. Thus in the early stages of all acute febrile diseases, and in gouty, rheumatic, and lithæmic conditions, the addition of a few drops of perchloride of iron to the saliva produces an abnormally vivid reaction; while in cases of chorea, lead poisoning, severe jaundice, dyspepsia, and marasmus, the salt may be entirely wanting, and fail to reappear until general improvement has set in (Fenwick).

(*h*) *The micro-organisms of the mouth.*—Numerous species of micro-organisms are constantly present in the buccal secretions and upon the moist surface of the mucous membrane of the mouth. Some of these are only occasionally encountered, and may be regarded as accidental; others appear to have their natural habitat in the buccal cavity. The non-pathogenetic organisms possess various properties; some curdle milk, others dissolve albumin; others, again, give rise to fermentation in solutions of glucose and lactose, or invert cane-sugar. Of the pathogenetic series the bacilli of cholera, typhoid, and diphtheria, the coccus of sputum septicæmia, and the diplococcus of pneumonia, are the most important. The saliva of healthy persons has the power of destroying or retarding the growth of the above-mentioned bacilli, but it constitutes an excellent medium for the development of the cocci (Sanarelli).

II. Gastric Digestion.—**Process of digestion in the stomach.**—The food, more or less triturated and incorporated with the saliva according to the efficiency of the dental apparatus, passes through the pharynx into the œsophagus, and thence into the stomach. The time occupied by this passage is so short that hardly any conversion of starch into sugar can take place. Arrived in the stomach the alimentary mass is subjected to a kneading process, which still further subdivides its particles and, with the addition of the gastric juice and swallowed fluids, helps to reduce it to a liquid state. Some part of this matter is absorbed directly through the walls of the stomach itself, but the larger part, when thoroughly prepared, passes out through the pylorus into the duodenum as *chyme*. The duration of the digestive process in the stomach varies with the individual, with the kind of food, with the activity of the gastric juice, and with the contractile power of the muscular walls of the organ. Speaking generally, the process is completed in from five to seven hours; though from the earliest period of gastric digestion small quantities of the softer and more easily digested portions of the food pass through the pyloric orifice, leaving the more solid residue to undergo further kneading and rotation, and to come under the influence of fresh supplies of gastric juice. The chyme is an acid fluid which passes from the pylorus into the

duodenum, there to be mingled with the alkaline secretions of the liver and pancreas. It consists of the various elements of the food, reduced to a soluble condition and altered by the gastric juice. These changes may be briefly summarised as follows: (*a*) The conversion of starch into sugar, begun in the mouth, is carried to a farther point; (*b*) Much of the proteid and albuminoid material is converted into peptones; (*c*) Fat, by the digestion of its cell walls, is partially emulsified; (*d*) Milk is curdled. The proper performance of gastric digestion may be disturbed mechanically by a delay in the reduction of the alimentary mass to a fluid state, by feebleness of the muscular movements, by any obstruction to the onward passage of the digested products through the pylorus—such as stenosis of its canal, or thickening of the mucous walls of the stomach itself; or by chemical changes, qualitative or quantitative, in the composition of the gastric juice, such as to impair or hinder the process of solution. But although the factors of delay are chiefly concerned in the production of gastric derangements, yet those which unduly hurry on the food before its complete digestion also play some part in causing intestinal indigestion. These consist of an unduly active peristalsis of the muscular walls, or a feebleness, qualitative or quantitative, of the gastric juice. The *gastric juice*, which can only be obtained mixed with mucus, through a gastric fistula, has an acid reaction and a slightly acid taste; its specific gravity, when at first secreted under excitation, is 1·001 to 1·003, but becomes higher as the process of secretion continues, when it also contains more corpuscular elements. It has the property of converting proteid bodies into diffusible peptones,—a property due to the combined action of an enzyme, called "pepsin," and hydrochloric acid, which latter appears either free or in combination with some organic body. In addition to pepsin, the gastric juice contains another ferment, namely, "rennin," a milk-curdling ferment. Besides these two ferments, and the acid which gives the secretion its normal reaction, the remaining constituents are of slight importance; they consist of mucus, fat, traces of organic acids resulting from digestion, and of certain salts, such as alkaline chlorides and earthy phosphates. Gastric juice exposed to the air, by its natural antiseptic properties, retains its activity for some considerable time without putrefaction. The secretion is only poured from the peptic glands under stimulation; when digestion is complete no more juice is secreted. From this it would seem that the process of gastric digestion is immediately under the control of the nervous system. It is probable, however, that the excitation is not due to a direct nervous action on the digestive glands, but rather to the muscular movements of the walls of the stomach. On this point it is well to recall Bence Jones's experiment, which shows at least how poorly the stomach is supplied with sensory nerves. This observer found that on taking a strong solution of tartaric acid he experienced a violent burning pain all down the gullet, which ceased to be felt in the region of the stomach till about two hours later, when acute pain arose about the region of the pylorus, owing, presumably, to the acid passing out into the duodenum. It may be, as

Heidenhain has suggested, that the secretion of the gastric glands occurs under mechanical irritation without any nervous influence at all. Of all stimuli applied to the secreting epithelium saliva is the most powerful; next to this, according to Claude Bernard, are dilute solutions of alcohol and of the alkaline carbonates. The act of deglutition also has the effect of increasing the flow of gastric juice, apparently by exciting those peristaltic movements of the stomach which are supposed to promote the activity of the peptic cells. For this reason dyspeptics have been advised to masticate small portions at a time and to swallow frequently.

Chemical examination of the contents of the stomach.—Of late years much attention has been given to the variations in the quantity and quality of the gastric secretion during the course of different diseases; and the knowledge which has been gained by the systematic examination of the process of digestion in disorders of the stomach has not only resulted in the distinction of several forms of dyspepsia which were formerly included under a single denomination, but has also brought to light certain facts of diagnostic and prognostic value. We shall now give a brief description of the principal methods which are in use at present for the chemical examination of the contents of the stomach.

The activity of the gastric secretion is at all times dependent to a great extent upon the quantity and quality of the food. Thus, in order to obtain results which are capable of comparison, it is necessary in every case to administer to the empty stomach a meal the composition of which is constant and accurately determined, and to evacuate the stomach after a definite interval of time. For this purpose several "test meals" have been recommended, of which the following are those most commonly employed:—

1. A breakfast (8 o'clock) consisting of 70 grammes of white bread and 300 c.c. of weak tea. The stomach is emptied after the lapse of an hour (Ewald).

2. A breakfast composed of 70 grammes of white bread and 500 c.c. of milk. The stomach is emptied after the lapse of two hours (Klemperer).

3. A lunch (11 o'clock) composed of 150 grammes of bread, 80 grammes of minced meat, and half a tumblerful of cold water. The stomach is emptied in two hours (Germain Sée).

4. A dinner (2 P.M.) composed of 100 c.c. of soup, 60 grammes of beef-steak, and 50 grammes of white bread. The stomach is emptied in five hours (Riegel).

Of all these the most convenient is Ewald's breakfast, since digestion is at its height at the end of an hour, when the contents of the stomach are comparatively clean and easily evacuated.

Extraction.—The stomach is usually emptied by means of a soft india-rubber tube, but it is sometimes convenient to direct the patient to tickle the fauces with his finger or a feather so as to excite reflex vomiting.

An hour after Ewald's meal about 30 c.c. of semi-digested material can usually be obtained. The mixture has a peculiar odour, like that exhaled by raw beef; but in cases where fermentation has been active in the stomach the smell is sour and disagreeable.

Filtration.—The first procedure is to filter the mixture through paper. The rate at which this can be accomplished varies in different cases; it is rapid and complete when the material is diffluent and contains a large quantity of hydrochloric acid, but extremely slow if much mucus be present. The filtrate consists of a limpid, yellow, and slightly opalescent fluid possessing a characteristic odour and capable of resisting decomposition for some time. Under pathological conditions, however, the colour may be green from admixture with bile; or red, or brown, if blood be present. In such cases also the odour may be sour (lactic acid), pungent (excess of hydrochloric acid, acetic acid), rancid (butyric), ethereal, saccharine, or offensive. The residue left upon the filter consists entirely of the materials which have escaped solution. If digestion has been active the amount is small, and the masses of bread appear swollen and gelatinous; but if the gastric secretion has been deficient the residue is considerable in amount, and its several constituents are little altered in appearance.

Reaction.—The filtrate always gives an acid reaction to litmus paper. This may depend upon the presence of (*a*) hydrochloric acid, free or combined; (*b*) organic acids; (*c*) acid salts. The presence of a free acid can easily be determined by testing the fluid with a piece of congo red paper, which, when brought into contact either with a mineral or an organic acid in an uncombined state, acquires a blue colour.

Total acidity.—The next step is to ascertain the total acidity of the filtered fluid. This is readily accomplished by triturating 10 c.c. with the decinormal solution of caustic soda (4 grammes per litre) until neutralisation has been effected. The result can either be expressed in terms of hydrochloric acid, or more simply by the number of cubic centimetres of the soda solution which were required to neutralise 100 c.c. of the filtrate. Thus, if 7·5 c.c. of the alkaline solution were sufficient to neutralise the 10 c.c. of the filtrate, then the total acidity of the gastric fluid is said to be 75.

Under normal conditions the total acidity, after Ewald's meal, varies between 50 and 65. An excessive acidity usually indicates an abnormal amount of hydrochloric acid; a diminished acidity generally depends upon a deficiency in the secretion of this acid.

Qualitative tests for the various free acids.—1. *Free hydrochloric acid.*—The presence of this mineral acid in a free state is most easily recognised by means of the reaction described by Günzburg. A few drops of the filtrate are mixed in a porcelain dish with an equal quantity of a solution composed of phloroglucine, 2 parts; vanillin, 1 part; absolute alcohol, 30 parts. If free hydrochloric acid is present, on the application of heat a ring of a crimson colour appears around the site of evaporation, the intensity of the colour being directly proportionate to the degree of

acidity. This test is an extremely delicate one, and is capable of detecting 0·002 per cent of (free) hydrochloric acid.

Boas recommends a test solution for hydrochloric acid composed of resorcine, 5 grammes; white sugar, 3 grammes; absolute alcohol, 100 c.c. The test is applied in the same manner as the former one, but to produce the red colour evaporation must be continued to dryness. As the dish cools the colour disappears. This reaction is sufficiently delicate to reveal the presence of 0·005 per cent of the acid.

Many other colour reagents vary in tint in the presence of a free mineral acid; but they also react in a similar manner, though in a less degree, to organic acids. Thus congo red, methyl violet, and benzopurpurine each become blue, while tropæoline (00) turns a carmine-red.

2. *Lactic acid.*—This acid is readily distinguished by means of the test proposed by Ueffelmann. To 20 c.c. of distilled water containing one drop of the tincture of the perchloride of iron, 10 c.c. of a 4 per cent solution of carbolic acid are added; the mixture, now of an amethyst blue colour, on the addition of lactic acid will immediately change to a canary yellow. This reaction is a very delicate one, but is hindered by the presence of phosphates, alcohol, glucose, or an excess of hydrochloric acid.

3. *Butyric acid.*—This acid produces a turbid brown precipitate with Ueffelmann's test, and possesses a characteristic rancid smell. If the filtrate be treated with an excess of ether, and the latter be separated and evaporated, the residue will contain the greater portion of the butyric acid which was originally present. If this residue be dissolved in water, and a few pieces of chloride of calcium added to it, butyric acid will separate in the form of oily droplets, which are readily recognised.

4. *Acetic acid.*—To detect the presence of this acid a small quantity of the filtrate is extracted with ether, the ether is evaporated, and the residue is dissolved in water and carefully neutralised with the decinormal solution of soda. If any acetate of sodium be present the addition of a drop of the tincture of perchloride of iron to the solution strikes a blood red colour.

Quantitative estimation of the various acids.—1. *Hydrochloric acid.*—This acid exists in the stomach both in a free state and also combined with various forms of proteid. It is necessary, therefore, to be able to estimate it in each condition.

The quantitative determination of *free* hydrochloric acid is most easily and quickly performed by means of a method introduced by Mintz. This method is based upon the fact that when a solution of soda is added to a mixture of mineral and organic acids the alkali will completely neutralise the former before entering into chemical combination with the latter. To put this method into practice, 10 c.c. of the filtered contents of the stomach are placed in a porcelain dish, and the decinormal solution of soda is allowed to fall into it, drop by drop, from a graduated burette. The mixture is repeatedly stirred with a glass rod, and from time to time a few drops are removed to a clean dish and tested with the phloro-

glucine vanillin solution. As soon as the latter fails to give the characteristic reaction it is obvious that all the free hydrochloric acid has been neutralised, and its percentage amount can readily be determined by multiplying the number of cubic centimetres of the soda solution which have been used by the number ·0365. It is wise to perform this estimation twice, and to take the mean of the two observations.

2. *Free and combined hydrochloric acid.*—Several methods have been devised for the determination of the total quantity of hydrochloric acid (free and combined) in the contents of the stomach (Hehner and Seemann, Sjoquist, Cahn and v. Mering, Leo, Hayem and Winter). The first method is the most convenient, and this only therefore will be described.

Method of Hehner and Seemann.—Ten cubic centimetres of the gastric filtrate are exactly neutralised with the decinormal solution of soda, and the quantity of the alkaline solution used for this purpose is carefully noted. By this procedure all the acids present in the filtrate, both free and combined, are converted into the corresponding salts of sodium, so that the mixture contains only lactates, butyrates, acetates, and chlorides of sodium. The mixture is now evaporated to dryness and the residue collected, placed in a platinum crucible, and carefully calcined. The incinerated material, which consists entirely of the carbonate and chloride of sodium, is dissolved in a sufficient quantity of distilled water, and to the solution is added an amount of the decinormal solution of hydrochloric acid exactly equal to that of the corresponding soda solution which was originally employed to neutralise the acid filtrate. A certain portion of this fresh acid is neutralised by the carbonate of sodium derived from the lactates and butyrates, while the surplus corresponds exactly to the amount of chloride of sodium present; that is, to the total amount of hydrochloric acid which was contained in the original 10 c.c. of the filtrate. In order to determine this quantitatively, the mixture is triturated with the standard solution of soda, and from the quantity of the latter required to effect neutralisation the amount of the mineral acid is calculated. Thus, if this second neutralisation has required 5 c.c. of the soda solution, the 10 c.c. of the filtrate must have contained $5 \times \cdot 00365 = 0\cdot 01825$ gramme of hydrochloric acid. In other words, the total quantity of hydrochloric acid present in the filtered contents of the stomach was 0·1825 per cent.

It has already been shown that by the method of Mintz the amount of free acid can be measured separately; in order, then, to determine the quantity of hydrochloric acid which exists in chemical combination with the proteids of the food, it is only necessary to subtract the result of the first examination from that of the second. Thus, if in the case abovementioned the free acid was found to be equal to 0·06 per cent, the amount of the combined acid must be 0·1225 per cent ($0\cdot 1825 - 0\cdot 06 = 0\cdot 1225$).

3. *Lactic acid.*—In order to estimate the quantity of this acid, 10 c.c. of the filtrate are boiled with a few drops of dilute sulphuric acid in order to coagulate any albumin which may be present. After filtration

the fluid is evaporated until it acquires a syrupy consistence; it is then mixed with 10 c.c. of distilled water, and evaporated to dryness in order to expel all the volatile acids. The residue is extracted with an excess of ether, and the ethereal solution is separated and carefully evaporated. The solid material which remains in the flask consists entirely of lactic acid. This is dissolved in water and triturated with the decinormal soda solution until neutralisation is complete. Then, since 1 c.c. of the soda solution corresponds to 0·009 gramme of lactic acid, it is only necessary to multiply this decimal by the number of cubic centimetres of the alkaline solution which were used in order to determine the amount of lactic acid contained in the original 10 c.c. of the filtrate (Cahn and v. Mering).

4. *Butyric and Acetic acids.*—The quantitative estimation of these acids has no practical value. However, it can be accomplished by the method described by Cahn and v. Mering, which has already been referred to.

Tests for the gastric ferments.—1. *Pepsin.*—The presence of the peptic ferment in the gastric contents is evidenced by the power of the filtrate to digest albumin. In order to determine this, a quantity of hydrochloric acid is added to 20 c.c. of the filtered contents of the stomach, in order to endow it with a degree of acidity equal to 0·2 per cent. The mixture is then placed in a flask containing several small cubes of egg albumin or shreds of fibrin, and set aside in a warm chamber. If the ferment is active the albumin will undergo solution, so that at the end of an hour its bulk will have appreciably diminished. The rapidity with which this change takes place affords a rough indication of the quantity of pepsin present.

2. *Rennet.*—This ferment is always present in a gastric juice which contains pepsin. To demonstrate its presence 10 c.c. of the filtrate are carefully neutralised and mixed with an equal quantity of sterile milk. After exposure to a temperature of 38° C. for half an hour the milk will have undergone coagulation, while the mixture still retains its neutral or alkaline reaction.

The soluble products of digestion.—In addition to the various acids and ferments which have been enumerated, the filtered contents of the stomach always contain some of the soluble products of digestion. Thus small quantities of achroo-dextrin, maltose, and dextrose, resulting from the action of ptyalin upon the starch, can usually be recognised; while the action of the gastric juice upon the albuminous constituents of the food is indicated by the presence of syntonin, propeptone and peptone. Minute quantities of fatty acids, arising from abnormal fermentation of the fats, may also be present.

The insoluble residue left upon the filter consists mainly of starch, cellulose, food débris of all kinds, fat, epithelium, mucus, and various forms of bacilli and micrococci.

Micro-organisms of the stomach.—Numerous bacilli and micrococci are habitually present in the stomach of healthy persons; they gain access to the organ either through the medium of the food or by re

gurgitation from the small intestine. Some of these are relatively inert, but the majority are certainly endowed with a specific fermentative action upon the various food-stuffs. Under normal conditions the growth and influence of these micro-organisms are controlled by the antiseptic properties of the hydrochloric acid of the gastric juice, which, in the proportion of 0·2 per cent, is capable of inhibiting most of the processes of fermentation. But in certain pathological states, either from a deficient secretion of the acid or an atonic condition of the walls of the stomach which permits stagnation of the food, bacterial fermentations are both numerous and active, and their products are responsible for most of the symptoms characteristic of dyspepsia.

Abelous was able to isolate at least sixteen distinct species of microbes from the gastric contents of healthy persons, including such diverse organisms as Sarcinae, B. lactis, B. pyocyaneus, B. subtilis, B. lactis erythrogenes, Vibrio rugula, B. amylobacter, B. megatherium, and many others. Several of these micro-organisms possess similar fermentative properties.

There are four principal forms of fermentation in the stomach :—1. *Lactic acid*, produced by the B. lactis and other varieties upon lactose and glucose. 2. *Butyric acid*, also produced by many different species of bacteria, but especially by the B. butyricus. This acid is formed from lactic acid, two molecules of the latter being transformed into one of the former, with the liberation of four volumes of hydrogen. 3. *Acetic acid.* The ordinary agent in the production of this acid is the mycoderma aceti, but it may also arise as a secondary product of lactic fermentation. 4. *Alcohol.* This is occasionally manufactured in dilated stomachs by the fermentation of sugar. The B. alcoholicus is introduced into the organ in fermented foods and new bread.

Acetone is also sometimes encountered, while the action of the various bacteria upon the nitrogenous constituents of the food occasionally gives rise to forms of peptone which possess highly poisonous properties.

The gases of the stomach.—In many cases the gaseous contents of the stomach consist entirely of atmospheric air which has been swallowed ; this air, however, tends to lose the greater part of its oxygen by absorption. Occasionally the eructated gas proves to be inflammable, and under these circumstances it usually consists of a mixture, in varying proportions, of oxygen, hydrogen, nitrogen, carbonic acid and marsh gas. In the majority of the cases of atony of the stomach, with fermentation, the gas consists of hydrogen and carbonic acid, with traces of nitrogen and oxygen. Sulphuretted hydrogen is produced by the decomposition of albumins, and is more commonly present in the intestine than in the stomach. It is, however, often eructated after large quantities of mineral waters or substances containing sulphides or sulphates have been taken.

The Physiology of digestion.—The introduction of food mixed with saliva at once excites the secretion of gastric juice, which continues

without intermission until gastric digestion is complete. But, although hydrochloric acid is secreted from the outset, the presence of free acid in the contents of the stomach can seldom be detected until after the lapse of a period of time which varies with the size and quality of the meal. Recent investigations have afforded a complete explanation of this peculiar circumstance. It has been shown that a strong affinity exists between all forms of albumin and free acids, each variety of proteid possessing a definite and constant acid equivalent. Thus, 100 grms. of milk are found to saturate or "fix" 0·298 grm. of hydrochloric acid, while the same quantity of meat requires 0·843 grm. of the acid; the resulting chemical compound is possessed of some stability, but, though acid in reaction, it never affords the colour reactions of a free acid. It may, therefore, be accepted that the contents of the stomach will appear to be devoid of free hydrochloric acid until the whole of the albuminous portions of the food have been completely saturated. This curious phenomenon helps to explain the constant presence of lactic acid during the early stages of normal digestion. Ewald and Boas were the first to show that, if the contents of the stomach are examined at short intervals after a test meal, the percentage of lactic acid undergoes a steady augmentation for the first twenty minutes; after this time it remains stationary for another twenty minutes, then rapidly diminishes and finally disappears. These observers also noted the significant fact that the diminution in the production of the organic acid coincides in point of time with the first appearance of free hydrochloric acid. Although this so-called antagonism between the mineral and organic acid is probably more apparent than real, since lactic acid is often introduced with the bread of the test meal, there can be little doubt that lactic acid fermentation habitually occurs during the preliminary stages of gastric digestion. The process, however, is kept within moderate bounds (0·1 per cent) by the timely appearance of free hydrochloric acid, which, as soon as it attains the proportion of 0·07 per cent, inhibits the further activity of the various micro-organisms.

Variations in the chemical constitution of the gastric juice.—*Hydrochloric Acid.*—Sudden and transient failure in the secretion of this acid occurs in cases of overloading of the stomach with fermentable substances (*embarras gastrique*). Free acid is generally absent when the stomach, especially in its pyloric portion, is the seat of carcinoma. The total output of acid is found to be constantly diminished in cases where the gastric mucous membrane is affected with catarrhal inflammation, cirrhosis, or fatty and lardaceous degenerations. It is also diminished in quantity in many instances of profound anæmia and neurasthenia.

An excessive secretion of hydrochloric acid, as the result of the introduction of food, is encountered in all cases of hyperacidity, whether the disorder be dependent upon direct irritation of the mucous membrane of the stomach by the ingesta, or upon indirect irritation from disease of the central nervous system. An abnormal degree of acidity is also present in most instances of hypersecretion and of ulcer.

The secretion of the *peptic ferment* is much more constant than that of the acid, and, even when the stomach is the seat of grave organic disease, the introduction of a dilute solution of hydrochloric acid will usually procure the elimination of pepsin. Indeed, it is only in those cases in which the mucous membrane has been destroyed almost completely by chronic inflammation that this ferment is absent from the gastric secretion.

The *rennet* ferment is the most constant of all the active constituents of the gastric juice, and only ceases to be secreted when the stomach is the seat of chronic and severe atrophy.

III. INTESTINAL DIGESTION.—Under normal conditions the digestive secretions of the stomach, and those which operate within the small intestine, are maintained in a state of physiological equilibrium, the entrance of the chyme into the duodenum provoking a reflex secretion of the pancreatic and biliary fluids to an extent exactly sufficient to neutralise the acid contents of the stomach, and to establish the degree of alkalinity in the mixture which is most suitable for the action of those ferments which complete the processes of digestion. A moderate increase in the acidity of the gastric contents is followed by an increased elimination of the bile and pancreatic secretion; while in certain functional disorders of the liver (and perhaps also of the pancreas) hyperacidity of the gastric juice is occasionally encountered—a phenomenon which may be explained upon the hypothesis that a more active stimulus than usual is required to call forth the tardy secretion of the biliary fluid. It follows from these facts that the digestion and subsequent absorption of food in the small intestine depend not only upon the integrity of the liver and pancreas, but even to a greater extent upon the due performance of the functions of the stomach. Intestinal indigestion may, therefore, originate either as a primary or a secondary disorder; but sooner or later in every case an aberration of function in one portion of the digestive tract is followed by derangement of the other parts.

Bile.—The composition and amount of the bile is naturally difficult to estimate in man, but it can be ascertained in the case of a long-established biliary fistula. Even in this instance, however, the gall-bladder may store up a considerable quantity of bile before it is poured into the common duct. Physiologists have often wondered why carnivorous animals should possess such a receptacle for storage, and why most herbivorous animals are without it. But, clinically, it appears that it is not an essential organ, as the gall-bladder is often found obliterated or obstructed after death, although no bad symptoms had been observed previously. Indeed, it has been remarked that when the cystic duct has been occluded by a calculus jaundice is of rare occurrence. It would seem, therefore, that the bile which flows direct from the hepatic duct is the best to make observations upon.

Quantity.—According to Professor Gamgee, who has made a critical analysis of the observations of different authors, those of Copeman

and Winston and of Mayo Robson possess an importance which cannot be attached to any previously recorded. From these researches we may conclude that the amount of bile secreted by the healthy human subject, when reabsorption of the bile from the intestine is prevented, varies from 1 pint to 1½ pint a day; whilst the solids excreted in the twenty-four hours amount to from 3·5 drachms to half an ounce.

General characters.—The bile as it flows from the hepatic duct differs from that retained in the gall-bladder. As it flows from the liver it is a thin transparent fluid of golden yellow colour, like yolk of egg, of bitter taste and alkaline reaction. In the gall-bladder and common duct it acquires a viscidity from the secretion of the glands of that portion of the mucous membrane, and often loses much of its alkaline reaction. Such bile is destitute of odour, and has a mawkish, bitter taste due to the bile acids. The specific gravity of the bile varies considerably according to different observations. Frerichs gives it as high as 1·03, whilst the more recent observations of Noel Paton and Balfour have recorded variations between 1·0054 and 1·008. This difference may depend on the fact that some of the earlier observers did not distinguish between a biliary fistula of the common duct and a fistula communicating directly with the gall-gladder; yet the specific gravity of the bile from the gall-bladder is always much higher than that obtained directly from the hepatic ducts. After being withdrawn from the body, bile has a brown-yellow tint, but by exposure to the air it gradually assumes a greenish colour, due to chemical alterations in the pigments.

Cholesterin ($C_{26}H_{44}O$) is a constant element of bile. According to Hoppe-Seyler 0·35, and to Frerichs 0·26 represents the amount in 100 parts, and later observers have not altered these figures. It is still doubtful whether to regard cholesterin, the chief fatty constituent of the bile, as formed in the liver or merely excreted from the blood. In forming an opinion on this point we must bear in mind that in certain forms of liver disease, particularly cirrhosis in which there is a great destruction of liver-cells, the termination is by coma, which has been attributed to the retention of cholesterin in the blood (cholesteræmia). We have no evidence, however, to show that cholesterin has any toxic influence. Krusenstern injected from 0·005 to 0·045 gramme of cholesterin daily into the veins of dogs, and found the animals unaffected. Pagès arrived at the same results. Looking at the question from a purely clinical side one would expect cholesteræmia in all cases of jaundice where there is considerable reabsorption of bile. If, as is generally held, the liver is the seat of the destruction of the blood corpuscles, which contain nearly all the cholesterin found in the blood in the normal state, we might naturally suppose that the cholesterin of the bile, reabsorbed in a free state into the blood, would rapidly induce cholesteræmia; but as a matter of fact the phenomena attendant on this condition are not common in obstructive jaundice, however complete; unless there be also considerable destruction of liver tissue and disease of the kidneys.

Again, in acute yellow atrophy, where there is rapid destruction of

the liver-cells but comparatively slight jaundice, the coma is preceded and attended by symptoms much resembling those witnessed when phosphorus is injected into the veins of animals, and in patients dying from acute diabetic coma (acetonæmia).

From these considerations it appears possible that the coma attendant upon hepatic disease associated with considerable destruction of liver-cells is not due to the accumulation of cholesterin, but to a general increase of the excretory matters in the blood; and this supposition is strengthened by the fact that the condition is never witnessed unless there be also some impairment of the renal functions.

Cholesterin is insoluble in water and dilute saline solutions. It is soluble in ether, from which it may be obtained in an amorphous state after evaporation. Treated with hot alcohol it is deposited on evaporation in characteristic rhombic plates, with notched edges, which float on water. Cholesterin is soluble in solutions of the salts of the bile acids, of soaps and neutral fats, which are probably the factors that keep it in solution in the bile. The chief tests for cholesterin are—(i.) Ready solubility in ether; (ii.) When heated with nitric acid, the mixture gives off yellow fumes of cholesteric acid, and the residue, on the addition of ammonia, acquires a brick-red colour. The latter reaction is somewhat similar to that which occurs with uric acid, and has led to deposits of cholesterin in the cardiac valves and vessels of the brain being mistaken for uratic concretions.

Lecithin is an organic fatty body which exists in the bile in the proportion of 0·25 per cent. In combination with cerebrin it was supposed to form protagon, but the recent researches of Gamgee seem to show that the latter is a definite chemical compound. According to this author, protagon, although it cannot be separated by the action of solvents into non-phosphorised cerebrin and a phosphorised body, can nevertheless by the use of caustic baryta be made to yield non-phosphorised substances. The fact that the bile contains so large a percentage of cholesterin and lecithin appears to denote that the liver plays an important part in elimination of the metabolic products of the nerve-centres. The other fatty constituents of the bile consist of saponifiable fats, especially the oleates and stearates. The latter possess a certain amount of interest, since in combination with the earthy bases they form the crust of many of the biliary calculi. In the case of the large solitary stone, which sometimes completely fills the gall-bladder, as much as two-thirds of the outer crust may be composed of stearate of lime.

Mucoid nucleo-albumin.—Bile owes its viscidity to this body, which it acquires during residence in the gall-bladder. Former investigations seemed to show that the material to which the viscidity was due was mucin, but subsequent observation proved that the reactions did not correspond with that body. Following up the subject, Paijkull, under the direction of Hammarsten, discovered that it belongs to the nucleo-albumins, and that in character it is like the nucleo-albumin discovered by Hammarsten in synovial fluid. The methods for the separation of this body are distinct

from those used for the precipitation of mucin, but as these are tedious, and of no use to the practical physician, the reader is referred to the excellent account of them given by Professor Gamgee. The fact, already stated, that this mucoid substance is derived from the gall-bladder and ducts is proved by treating an aqueous extract of the secretion of these structures with acetic acid, when a body is obtained which in all respects resembles the nucleo-albumin obtained from the bile itself. Ordinary mucin exists, nevertheless, in small quantities in the bile, and is probably derived from the hepatic ducts.

Bile acids.—In carnivorous animals the taurocholic acid is often the sole bile acid, whilst in the herbivora the glycocholic acid is met with almost exclusively. In man the glycocholic acid is the chief constituent, taurocholic acid being usually present in traces only.

Glycocholic acid ($C_{26}H_{43}NO_6$). Several methods of isolating this substance are described in the physiological text-books. Gamgee recommends Hüfner's method as yielding the best results, when successful; but, as he points out, an excess of taurocholic acid in the bile prevents the precipitation of the glycocholic acid. This, however, should constitute no practical objection to its use in separating glycocholic acid from human bile, in which taurocholic acid is present in small quantities only. Glycocholic acid separates out in fine transparent needles. These are highly insoluble in cold water (1-300), but less so in hot water (1-102). They are almost wholly insoluble in ether, but are freely soluble in alcohol and glycerine. They have an acid reaction and a bitter-sweet taste. Both the acid and its salts are dextrogyrous. Sodium glycocholate may be obtained by dissolving the acid in a solution of sodium carbonate, then evaporating to dryness, dissolving the residue in absolute alcohol, and adding anhydrous ether, when the salt separates out in fine transparent needles which are readily soluble in alcohol and in water. An aqueous solution is precipitated by neutral lead acetate, and its solution has a partial action on saponifiable soups. The test for glycocholic acid and its derivatives has long been known as "Pettenkofer's," the principle of which is the development of a fine purple colour on the admixture of cane-sugar and the very gradual addition of strong sulphuric acid. Several modifications have since been introduced to prevent the carbonising of the sugar, which is very apt to occur if the sulphuric acid be added too hurriedly, or the temperature allowed to rise above 70° C. Thus Dreschel has proposed the use of phosphoric acid instead of sulphuric, and Mylius has suggested the adoption of furfurol in water instead of sugar; furfurol being a product of the action of sulphuric acid on sugar. A very useful modification of Pettenkofer's test was proposed by Mr. Francis, formerly demonstrator of chemistry at Charing Cross Hospital; but, as he did not publish it, it is but little known. A few pieces of glucose are taken and dried thoroughly (when anhydrous they are to be placed in a well-dried stoppered bottle, and kept in the drying-chamber till wanted); then a small powdered fragment is placed in the bottom of a test-tube, and strong sulphuric acid (1 c.c.) added very gradually; on the surface of this

a small quantity of urine is poured, when the zone of contact of the two fluids will become opaque, and then a bright red gradually deepening into purple. If large quantities are required for clinical purposes the glucose and sulphuric acid may be mixed together and kept in a stoppered bottle in a cool place. The mixture forms sulpho-saccharic acid, of a pale straw tint, which gradually blackens; but, if kept at a low temperature and in the dark, it may be preserved for three or four days. As many other substances — notably morphia, albumin, and the phenols — give almost similar reactions with Pettenkofer's test, it is useful to employ the spectroscope for the identification of glycocholic acid and its derivatives. *Taurocholic acid* ($C_{26}H_{45}NSO_7$) occurs only in small quantities in human bile, and is sometimes absent; but in the dog it is the only acid present: for experimental purposes, therefore, recourse must be had to the bile of this animal. For the preparation of taurocholic acid the bile must be freed from all traces of glycocholic acid by precipitation with neutral lead acetate, after which the taurocholic acid is precipitated by means of a solution of ammonia and basic lead acetate in the form of lead taurocholate. This precipitate is well washed, boiled with absolute alcohol, filtered, and then treated with H_2S. The sulphide of lead is separated by filtration, the filtrate concentrated and then mixed with an excess of ether. The final precipitate, which is syrupy at first, is gradually converted into a mass of needle-shaped crystals (Hoppe-Seyler). Taurocholic acid is less stable than the glycocholic; its salts are less unstable, but still are not so stable as the glycocholates. Although taurocholic acid is intensely acid its alkaline salts are neutral, and are soluble in water and alcohol; their aqueous solutions foam like soap. Acidified with dilute hydrochloric acid solutions of the taurocholic acid and its salts precipitate albumin, acid albumin, and parapeptone; whilst the albumoses and peptones precipitate the acid. Both the bile acids yield by-products which, however, are not important clinically; the most interesting of them is *Cholalic acid*, which is not found in normal bile, but in bile that has undergone decomposition; it is said to occur in small quantities in the small and large intestines.

The bile pigments must be regarded as middle products in a series of reducing processes which convert the blood pigment into the colouring matter of the urine. Fresh bile owes its golden yellow colour to one of its constituents, bilirubin, which can be obtained in a pure state in the form of an orange-yellow powder endowed with a characteristic spectrum. By exposure to the air or to the influence of other oxidising agents, bilirubin can be converted through several intermediate stages (biliprasin, bilifuscin) into a green substance termed biliverdin, the spectrum of which is quite distinct from that of the original material. The presence of bile pigment is easily recognised by the display of colours which ensues upon the addition of fuming nitric acid (Gmelin). So far as our present knowledge goes, the pigments of the bile exert no influence whatever upon the processes of digestion.

Salts.—Besides the large quantity of alkaline sodium salts that are

combined with the bile acids, bile contains a considerable quantity of sodium chloride and sodium phosphate, traces of calcium and magnesium phosphate, and also traces of copper. Iron in variable quantities is always found in bile ash, and this fact is rendered more important by the researches of Dr. William Hunter, who discovered small deposits of iron in the biliary cells in diseases which lead to rapid destruction of the blood, such as pernicious anæmia, etc. [*vide* article "Liver," vol. iv.]

The Pancreas.—The normal secretion of the pancreas is a clear, viscid fluid which possesses an alkaline reaction, and froths when shaken. It contains very few structural elements, or none. Its most important constituents are alkali-albumin, small quantities of fats and soaps, a trace of leucin and tyrosin, and a comparatively large amount (1 per cent) of carbonate of sodium.

Action upon the various food-stuffs.—On starch, whether raw or boiled, the pancreatic juice exerts a specific action, converting it into erythro-dextrin and achroo-dextrin, and finally into maltose and glucose. This diastatic property depends upon the presence in the secretion of a special ferment closely allied to ptyalin, and named amylopsin. From the juice, or by the glycerine method from the fresh gland itself, a proteolytic ferment named trypsin can be isolated; this in the presence of carbonate of sodium rapidly reduces albuminous substances to a soluble state. The first effect of the ferment is to convert the proteid into the form of alkali-albumin, the final stage of its chemical action being the production of peptone. Some of the intermediate compounds are capable of being changed by the continued action of the secretion into a series of by-products of which the most important are leucin, tyrosin, indol, and skatol.

The pancreatic juice possesses also the power of coagulating milk; it contains a special ferment for this purpose, which can be chemically separated from trypsin. It differs from the latter in that it is able to exert its specific action in the presence of an acid.

The action of the secretion of the pancreas upon oils and fats is to produce emulsification and finally saponification, and in this way to promote the absorption of these substances by the intestinal mucous membrane.

I. *Primary intestinal dyspepsia.*—This may arise from (*a*) changes in the quantity or quality of the bile; (*b*) an insufficient secretion of the pancreatic juice; (*c*) disease of the mucous membrane of the small intestine.

(*a*) Obstruction to the flow of bile into the duodenum is usually accompanied by certain subjective symptoms which are included under the name dyspepsia. They comprise a sense of fulness or discomfort at the epigastrium after a meal, nausea, acidity, and flatulence. The bowels are confined, and the stools are hard and clay-coloured. These symptoms probably owe their origin to an excessive fermentation of the food consequent upon the loss of the antiseptic functions of the bile, and on the stagnation of the contents of the intestine from the lack of the same secretion.

Although it is true that persons with biliary fistula may live for many years without suffering any deterioration of the general health,

careful examination shows that the absorption of fat in these cases is considerably affected, as much as 55 to 78 per cent of the total quantity ingested being sometimes eliminated with the fæces. Very little is known concerning the alterations which take place in the quality of the biliary secretion. It is certain, however, that many substances which would act injuriously upon the organism are eliminated by the liver, and are thus discharged by the bowel. It has also been suggested that in some cases where the bile contains toxic material the secretion may irritate the duodenum directly and lead to catarrh of the mucous membrane, and finally to ulceration.

(*b*) Carcinomatous disease of the head of the pancreas, obstruction of its duct by a stone, or atrophy of its glandular tissue, are all associated with a diminution or absence of the pancreatic juice. Under these conditions it is sometimes possible to detect an excess of fat or of crystals of fatty acids in the fæces. According to the researches of Abelmann upon the digestion of food-stuffs after excision of the pancreas, starch is readily absorbed by the intestine, though only a portion of it is converted into sugar; about 44 per cent of the albumins is absorbed during the same period of time; while the greater portion of the ingested fat is split up into fatty acids which appear in the dejecta. Emulsions of fat in the form of soap and gum emulsions are not absorbed. These figures are all much more favourable when the pancreas is only extirpated in part, even though the flow of the secretion into the intestine be entirely prevented. It must be accepted, then, that the active agents of the pancreatic juice are capable on emergency of undergoing vicarious excretion with the other intestinal fluids. Total excision of the gland in dogs is usually followed by glycosuria, but the exact significance of this fact has not yet been definitely settled. [*Vide* art. "Diabetes Mellitus," p. 208.]

(*c*) Diseases of the small intestine may be accompanied by dyspepsia if the lesion deprives the mucous membrane of its power of absorption. This is chiefly the case when the lining membrane of the bowel is affected with a chronic form of catarrh leading to atrophy, a condition particularly apt to occur in children placed under unfavourable dietetic conditions, and in persons affected with chronic pulmonary tuberculosis (Fenwick).

II. *Secondary intestinal dyspepsia.* — Any form of gastric dyspepsia may give rise to derangement of the intestinal functions. (*a*) In cases of hyperacidity of the gastric juice the abnormally acid state of the chyme may completely neutralise the alkaline secretions which it encounters in the duodenum and thus put a stop to the digestive functions of the pancreatic juice. The unaltered starch which is thus allowed to accumulate undergoes acid fermentation under the influence of the various micro-organisms which inhabit this portion of the bowel. The absorption of fat is also delayed. The loss of the laxative properties of the bile increases the existing tendency to constipation, and at a later period the irritation due to the abnormally acid contents of the intestine gives rise to chronic

catarrh of the mucous membrane of the colon with dysenteric symptoms. (*b*) A deficient secretion of the gastric juice also exerts a detrimental effect upon the course of intestinal digestion, owing to the fact that fermentation of the food takes place in the stomach, and the duodenum consequently receives a large quantity of an extremely acid and undigested form of chyme. The ultimate result is the same as in the previous case. (*c*) Atony of the stomach, with loss of power in its muscular walls, leads of necessity to stagnation of the food, acid fermentation, and, finally, to the same effects upon duodenal digestion which occur in cases of hyper- or subacidity.

Fæces.—The average daily quantity of fæces passed by a healthy man is about five ounces, and constitutes nearly one-seventh of the total quantity of the food ingested. In early infancy this proportion is somewhat greater, but it declines considerably with the approach of old age. Diseases of the stomach associated with vomiting and loss of appetite are accompanied by a marked diminution in the daily output of fæces; but those disorders of the digestive system which are characterised by irritation of the intestinal tract are usually accompanied by the frequent passage of stools in which the liquid element predominates.

Consistence.—During the first six months of life the stools present a peculiar homogeneous, butter-like consistence; at a later period they assume the solid and cylindrical form of excrement which results from the use of a mixed diet. Increased fluidity of the stools rarely arises from insufficient absorption of the ingested fluids (lienteric diarrhœa); more often it is the result of an excessive secretion by the mucous membrane of the intestine due to catarrhal inflammation, ulceration, or the administration of purgative drugs. An abnormally dry or tenacious state of the dejecta, on the other hand, may result from the undue absorption of the fluid contents of the bowel during its protracted residence in the intestine (constipation), or from a lack of secretion on the part of the mucous membrane of the gut, which may be due to the astringency of such drugs as opium, bismuth, lead, and chalk. Sometimes the fæcal masses present a uniform coating of thick mucus, or the excreta may consist almost entirely of this material. These abnormal appearances indicate a catarrhal condition of the mucous membrane of the large intestine, and therefore frequently accompany functional disorders of the digestive organs, especially in early life. Finally, it may be observed that in those forms of dyspepsia in which excessive fermentation occurs among the contents of the gastro-intestinal tract, the stools may present a pultaceous, frothy, or yeast-like appearance.

The reaction of healthy stools is usually acid, but its degree depends to a great extent upon the nature of the food. Increased acidity accompanies an excessive secretion of the gastric juice (hypersecretion, hyperacidity), as well as those numerous forms of disordered digestion which are associated with fermentative changes in the contents of the intestine. Diminished acidity of the stools is sometimes observed in the diarrhœa of infants, and in that which accompanies cholera and typhoid fever.

Colour.—The stools derive their yellow colour from certain pigments manufactured from the bile by a process of oxidation (stercobilin, hydrobilirubin). The intensity of the colour varies directly with the activity of the biliary secretion, and with the length of time the undigested portions of the food have been retained in the intestine. Pale or colourless fæces, on the other hand, indicate a deficient entry of bile into the bowel; and accordingly gray, white, or putty-like motions usually accompany those functional and organic diseases of the liver which are associated with a diminished activity of the hepatic cells, as well as those diseases which mechanically obstruct the bile-ducts. Green stools are frequently observed as a result of digestive disorders in infancy. The peculiar colour has been ascribed to the presence of a chromogenetic bacillus (Hayem, Le Sage); but it appears to depend rather upon an alkaline decomposition of the contents of the small intestine (Pfeiffer).

Black coloration of the fæces occasionally arises when altered blood is present (melæna); but it is more frequently observed as the result of the administration of such drugs as bismuth, iron, and charcoal. Certain articles of diet and a few medicinal remedies are also apt to impart their characteristic colour to the motions; thus claret and logwood produce reddish or violet dejecta; coffee and beef tea a brownish, and rhubarb and santonine a bright yellow tint.

Odour.—The fæcal odour depends upon the presence of certain volatile products of food decomposition (skatol, indol). It is consequently exaggerated in all cases of intestinal indigestion associated with excessive putrefaction of the contents of the bowel, whether this depend upon a primary disorder of the stomach, liver, or pancreas. The sour smell of the motions in infancy is due to the presence of organic acids derived from fermentation of the milk sugar.

Chemistry.—Healthy fæces consist approximately of about 25 per cent of solids and 75 per cent of water; but the relative proportion of these two constituents varies considerably both in health and disease. Among the normal constituents must be reckoned such products of pancreatic digestion as leucin, tyrosin, indol and skatol; butyric, proprionic, lactic and valerianic acids; alkaline phosphates and other salts; neutral fats and soluble soaps; urobilin and stercobilin, and small quantities of ferments the chemical action of which is similar to that of pepsin and ptyalin.

Microscopic examination of the stools seldom affords any trustworthy indication of the nature of the digestive derangement. It may be stated, however, that an excess of starch in the fæces indicates a defective action of the diastatic ferments secreted by the salivary glands and pancreas; while an undue amount of undigested proteids points to the functional failure of the gastric and pancreatic secretions. Blood, pus, mucus, foreign bodies, entozoa and bacteria if they occur in the stools are easily recognisable.

CHARLES HENRY RALFE.
W. SOLTAU FENWICK.

REFERENCES

1. BERNARD. *Leçons de physiologie expérimentale*, vol. ii. 1856.—2. BOAS. *Allgemeine Diagnostic und Therapie der Magenkrankheiten*, 1890.—3. CAHN and VON MERING. "Ueber die Säuren des Gesunden und Kranken Magens," *Deut. Arch. f. klin. Med.* Bd. xxxix. 3 and 4.—4. COHNHEIM. *Lectures on Pathology*, vol. iii. pp. 829, 830, 832.—5. COPEMAN and WINSTON. "Observations on Human Bile," *Journ. Physiol.* vol. x. p. 213.—6. DRESCHEL. "Eine Modification der Pettenkofer'schen Reaction," *Journ. f. prakt. Chemie*, vol. xxiv. p. 44.—7. EWALD. *Lectures on Digestion*, vol. i. p. 71.—8. FENWICK, SAMUEL. *Saliva as a Test for Diseases of the Liver*, 1889.—9. FENWICK, SOLTAU. *The Dyspepsia of Phthisis*, 1894.—10. *Idem. Disorders of Digestion in Childhood*, 1897.—11. GAMGEE. *Physiological Chemistry*, vol. ii. pp. 266-425.—12. GÜNSBURG. "Ein neues Reagens für dem Nachweis freier Salzsäure," *Centralbl. f. klin. Med.* 1887, 40.—13. HAMMARSTEN. "Studien über Mucin und Mucin-ähnliche Substanzen," *Pflüger's Archiv*, vol. xxxvi. p. 373.—14 HAYEM and WINTER. *Du Chemisme stomacal*, 1891.—15. HAIDENHAIN. "Physiologie der Absonderungsvorgänge," Hermann's *Handbuch*, vol. v. p. 55.—16. HOPPE-SEYLER. *Physiologische Chemie*, 1878, p. 308.—17. KLEMPERER. "Zur chemischen Diagnostic der Magenkrankheiten," *Zeit. f. klin. Med.* Bd. xiv. p. 156.—18. LEA. "A Comparative Study of Artificial and Natural Digestion," *Journ. Physiol.* vol. xi. p. 234.—19. LEO. "Ueber die Function des normalen und kranken Magens," *Berl. klin. Woch.* 1889, p. 227.—20. MINTZ. "Eine einfache Methode zur quantitativen Bestimmung der freien Salzsäure im Mageninhalt," *Wien. klin. Woch.* 1889, 20.—21. MYLIUS. "Zur Kenntniss der Pettenkofer'schen Reaction," *Zeit. f. phys. Chemie*, vol. xi. p. 492.—22. PAIJKULL. "Ueber die Schleimsubstanz der Galle," *ibid.* Bd. xii. p. 196.—23. PATON. "Observations on the Flow and Composition of the Bile in Man," *Rep. Coll. Physic. Edinb.* vol. iii. p. 191.—24. ROBERTS. *Diet and Digestion*, 1897, p. 68.—25. ROBSON. "Observations on the Secretion of Bile," *Proc. Royal Soc.* vol. xlvii. p. 499.—26. SJOQUIST. "Eine neue Methode freie Salzsäure im Mageninhalt zu bestimmen," *Zeit. f. phys. Chemie*, Bd. xiii. p. 1.—27. UEFFELMANN. "Ueber die Methode der Untersuchung des Mageninhaltes und freie Säuren," *Deut. Arch. f. klin. Med.* Bd. xxvi. p. 431.

C. H. R.
W. S. F.

GENERAL PATHOLOGY OF SECRETION

SECRETION is one of the fundamental phenomena exhibited by living protoplasm. In secretion the cell protoplasm elaborates certain substances, and these are subsequently extruded from the cells. In the higher forms of life secretory processes are specialised so that the function of secretion becomes localised in certain cell aggregates known as "glands." The substances elaborated by the activity of gland cells are usually, but not always, extruded from the gland by solution or suspension in water; hence the discharge of water becomes the most obvious and one of the most important of the conditions of secretion. In a true secretion the gland elaborates the peculiar elements contained in the secretion; it does not simply remove them from the blood. The salivary cells make ptyalin and mucin, and the gastric cells pepsin; the kidney cells, on the other hand, simply remove urea from the blood: thus the elimination of urea by the kidney is not a true secretion, but rather an excretion. Other constituents of the urine, however, such as hippuric acid, are made by the renal cells. Some physiologists consider that a fundamental distinction between a secretion and an excretion lies in the fact that the latter is passed to the exterior, without playing any further part in the economy;

whereas the former, even if ultimately voided, fulfils some more or less important function meanwhile. A more vital distinction, however, is that previously mentioned, namely, that in a secretion the essential constituent is elaborated by the gland cell; whereas in the excretion it is simply removed by the gland cell from the blood or lymph circulating in the gland. Glands may, therefore, secrete or excrete; or again the same gland may do both. This double function is more especially seen in pathological conditions, when secretory glands not uncommonly excrete certain more or less toxic and abnormal constituents present in the blood plasma. The biliary function of the liver is in part a secretion and in part an excretion; some of the characteristic constituents of the bile are elaborated by the liver itself, and other constituents of the bile, the pigments, for example, being, as it would seem, more or less effete products of metabolism, and of no further use to the economy, are thrown out.

Glands are generally provided with ducts by means of which the secretions or excretions are passed either to the organ where they are used, or to the outside. Certain glands, however, such as the thyroid, suprarenal, and pituitary, have no ducts; and the function of these ductless glands has always been a difficult problem. At one time their function was supposed to be the destruction of certain more or less toxic substances produced by the activity of the tissues, and this view is still held by some physiologists; by others it is held that these ductless glands also produce substances necessary either to certain tissues, or to the body at large, for the maintenance of physiological equilibrium. This function has been called an "internal secretion," which denotes that the specific material elaborated by the gland is distributed to the tissues of the body, not by ducts but by the blood-vessels or lymphatics of the gland. It has been definitely proved that certain glands, such as the thyroid and suprarenals, elaborate peculiar substances necessary for the maintenance of life; these glands at any rate have internal secretions. Further, it is highly probable that many glands with external secretions have internal secretions also; the glycogenic and sugar-forming functions of the liver supply an instance in point. The formation of sugar in the liver, and the subsequent distribution of this sugar to the body through the hepatic vein, is really an illustration of an internal secretion. The material elaborated by the suprarenals is mainly of service to the vascular system; that produced by the thyroid is probably destined for the nervous system, and it would seem that the sugar produced by the liver is mainly required by the muscular system. Some physiologists do not consider the liver a sugar-forming, but rather a sugar-stopping organ. If this latter view were confirmed, the liver could no longer be regarded as having an internal secretion.

Certain glands have no external, but an internal secretion only; the thyroid and suprarenal bodies afford illustrations of this variety. Other glands, such as the liver, the pancreas, and perhaps the kidneys, have both external and internal secretions. Finally, other glands, like the salivary glands, have external secretions only.

EXTERNAL SECRETIONS.—The following processes occur during the complete act of external secretion:—

(i.) The gradual elaboration and storage in the secreting cell of the specific organic constituents of the secretion, usually in the form of a zymogen; a zymogen is the antecedent of the ferment of the secretion—pepsinogen, trypsinogen, for example.

(ii.) The rapid conversion of this zymogen into the actual substance found in the secretion—into mucin or trypsin, for example. This conversion occurs suddenly during the act of secretion.

(iii.) The sudden discharge of a considerable amount of fluid, the amount varying in the several glands; it is large in the case of the salivary and gastric glands, moderate in the case of the liver, small in the case of the pancreas.

(iv.) The reconstruction of the protoplasm of the gland cells, and not infrequently, as in the salivary and mammary glands, the replacement of used-up cells by freshly-growing and active ones.

Thus there is a slow elaboration of the specific constituents of the secretion, and a slow building up of the gland cells anew, interrupted by a sudden discharge of fluid, together with an equally sudden conversion of the zymogen into the active and essential constituent of the secretion. In some cases the cells forming the gland actually break down into the constituents of the secretion; this is well seen in the mucous salivary glands and in the mammary glands. In other glands—as in the serous salivary glands, the gastric glands, or the pancreas—the zymogen is seen to accumulate in the form of granules, and after activity to remain only in the inner or lumen end of the cells. The formation of these granules can be shown in certain cases to be under the influence of the nervous system. Stimulation of the cervical sympathetic causes these granules to be formed in abundance in the cells of the parotid, and the saliva subsequently secreted is abnormally rich in organic constituents.

These granules are composed of a zymogen, and not of a ferment; this is shown by the fact that a fresh extract made from a gland in full activity, such as the stomach or pancreas during digestion, contains no active ferment such as pepsin or trypsin. If the stomach be extracted after treatment with dilute hydrochloric acid, the extract is found to be powerfully proteolytic, and to contain an abundance of ferment. Extracts of fresh pancreas are possessed of but little digestive power, even when made from the pancreas of an animal killed during digestion; if, however, the pancreas be treated with oxidising agents the extract then obtained is a powerful digester of proteids.

The elimination of water by different glands during secretion is probably effected by various means. In the case of the salivary glands it is very abundant, and is independent of the state of the circulation; but it is closely dependent upon the nervous system, and especially on the activity of certain gland nerves. In the kidney, on the other hand, the amount of water excreted is very closely dependent upon the rate of the circulation through the renal vessels, and there is as yet no proof of

any nervous control of the secretion other than that of the vaso-motor nerves which govern the renal circulation. In the cases of the stomach, intestines, and pancreas there is no clear evidence that the excitation of any nerve will cause the secretion to flow from these glands; although there is reason to suppose that their secretion is controlled in some way by the nervous system. The secretory activity of a gland is usually accompanied by a dilatation of its blood-vessels, and this is especially well seen in the salivary glands; yet even here this vascular dilatation is not an invariable accompaniment of the secretory act, and there is abundant proof that the discharge of the water during secretion is not a filtration dependent upon the local vascular dilatation. There is no adequate explanation of the mechanism by means of which the salivary cell is enabled to secrete these large quantities of water as a result of nerve excitation. In certain glands the individual cells can be seen to swell up largely at the moment of excitation, and it may be that the cell substance becomes suddenly hygroscopic, quickly imbibes fluid from the lymphatics, and as suddenly liberates it on the outward side.

Gland-nerves are concerned not only in the secretion of water, but, in the case of the salivary glands at any rate, they also contain fibres that exert an influence on the elaboration of the organic constituents of the secretion. Gland-nerves are therefore held to contain at least two sets of fibres: one set the so-called "secretory" fibres, most abundant in the cerebral nerves, such as the chorda tympani and nerve of Jacobson, the excitation of which causes the secretion of water; the other set the so-called "trophic" fibres, most abundant in the sympathetic, the excitation of which causes the gland cell to elaborate the organic and probably also the inorganic constituents of the secretion. These two separate processes of secretion, the elimination of water and the formation of the specific constituents, occur not only at different times, but also, in the case of the salivary glands at any rate, under the control of different nerve-fibres.

Division of the cerebral nerves supplying the salivary glands is followed by a slow atrophy of the gland or glands, and the appearance of a slow, continuous secretion known as the "paralytic secretion." This continuous secretion is not produced by division of the sympathetic nerves; the cerebral nerve must be divided, whether the sympathetic nerve be divided or not is immaterial.

A "paralytic secretion," following the division of the nerves distributed to the pancreas and intestine respectively, has also been described.

In the case of the salivary glands the paralytic secretion is accompanied by an atrophy of the gland; hence it is probable that the reconstruction of the gland protoplasm is also under the control of the nervous system.

The nervous mechanism of secretion is most obvious in the case of the salivary glands; in most of the other glands the evidence of this mechanism is as yet very imperfect. Normally the salivary secretion, like most secretions, is evoked by reflex excitation, the afferent nerves being the lingual and glosso-pharyngeal, and the stimulus the presence of

food or other substances in the mouth; dilute acids applied to the mucous membrane of the mouth and tongue are particularly efficacious. Dilute acids, if applied to the mucous membrane of the duodenum, are said likewise to cause a discharge of bile from the gall-bladder into the intestine. In the case of other glands, as of the stomach and pancreas, but more especially of the stomach, it is probable that secretion can be produced reflexly, notwithstanding the absence of direct proof of the existence of secretory nerves. In the article on the "Pathology of Digestion" it is stated that the presence of food in the mouth will cause a flow of gastric juice when, by previous ligature of the œsophagus, or by dividing the œsophagus and stitching the lower end of the upper segment to a wound in the neck, the masticated food is prevented from reaching the stomach: the sight of food will cause a secretion both of saliva and of gastric juice, the latter after ligature of the œsophagus to prevent the entry of saliva into the stomach. In some of these cases of secretion apparently reflex, it is possible that the secretion is really due to the excitation of the stomach by certain chemical stimuli reaching the organ through its blood-vessels, and derived from absorption of the food in the mouth or œsophagus. Although gastric secretion is readily produced by mechanical excitation of the stomach, a far greater amount of secretion is produced by the action of digestible than of indigestible substances [*vide* art. "General Pathology of Digestion," p. 287].

Secretion can also be produced by excitation of the higher portions of the nervous system; puncture of the medulla causes not only an increased urinary flow, but also a secretion of pancreatic juice; it is not certain that either is dependent solely upon the vascular effects produced by the puncture. Further, the excitation of the cerebral cortex in the epileptic paroxysm, when produced experimentally, as for instance by absinthe, produces a notable flow of saliva.

Secretions are not only evoked by direct or indirect excitation of the nervous system, but may also be arrested or inhibited. The salivary secretion can be inhibited by fear, and the pancreatic secretion can be arrested by the excitation of the central end of an afferent nerve. Experimentally, the formation of urine by the kidney is sometimes entirely stopped by tying a canula into the ureter; and, clinically, complete suppression is not uncommonly seen as a result of sundry injuries not directly involving the urinary apparatus. It is difficult to believe that in these instances the total suppression of urine is due entirely to disturbance of the circulation.

The nervous regulation of the activity of the sweat-glands is of interest; although the sweat is practically an excretion, yet the nervous mechanism of these glands is as perfect as in the case of the salivary glands. The sweat-nerves leave the spinal cord by the anterior roots in the dorsal and upper lumbar regions, and then enter the sympathetic system to be distributed to the body at large in the sympathetic fibres. In some cases, as in the head, face, and neck, these nerve-fibres are found in the main sympathetic nerves of the part; in other cases, as in

the limbs, the sweat-nerves are found in the nerves forming the limb plexuses; but these plexuses have received them by way of the communications existing between themselves and the sympathetic system. The sweat-nerves, although ultimately in the limb-nerves, do not leave the cervical or lumbar regions of the cord in the anterior roots of these nerves. These sweat-nerves have a very similar course to that of the vaso-motor nerves, but they are quite separate and distinct from them. The excitation of a sweat-nerve causes a secretion of sweat, and after the division of such a nerve the area of skin supplied by it sweats no longer. Usually the secretion of sweat is brought about through the nervous system either reflexly, or by emotions, or by the chemical action of certain substances on the nerve-centres. External warmth does not cause sweating by any direct action on the glands, but indirectly through the nervous system.

Uses of external secretions and of excretions.—Excretions remove more or less harmful substances from the blood and tissues. They also eliminate water, and so help to maintain the amount of water present in the tissues at the normal standard. Thus indirectly they aid in the maintenance of the body temperature at its proper mean. Most external secretions are concerned in digestion either as lubricants, such as saliva, or as digestants, such as saliva, gastric and pancreatic juice; sometimes, in addition to this, secretions have some special action, such as the alleged antiseptic action of the hydrochloric acid of the gastric juice. The substances present in some excretions are removed sometimes by what seems to be a roundabout channel; the urine, for instance, contains appreciable quantities of various aromatic bodies and conjugated acids that are apparently formed in the alimentary canal. It is quite possible, if the excretory activity of the kidney were greatly diminished, for these and other bodies, perhaps toxic in nature, to accumulate in the intestinal tract. Again, there is a distinct selective action on the part of different glands, as abnormal constituents present in the blood are not removed by all excretions alike; thus in cases of jaundice bile pigments are more especially excreted in the urine, and not at all in the saliva.

Some of the digestive juices are secreted in very large quantities; the secretion of gastric juice has been estimated at as much as seven litres a day: the great bulk of the fluid, however, is reabsorbed and not lost to the economy. The amount of fluid which daily passes into the alimentary canal and is again reabsorbed must be very large, and the daily quantities ingested and excreted form but a small fraction of the total amount. Of the solid constituents of these secretions also many are reabsorbed, and if, owing to a fistulous issue,—pancreatic or biliary, for example,—this be impossible, the percentage of solids in the secretion affected diminishes greatly; in the case of pancreatic or biliary fistula the percentage of solids may fall from an initial amount of 12 to 14 per cent to a final amount of 2 to 4 per cent. All the organic constituents, however, are not reabsorbed, since the urine contains small quantities of the various digestive ferments; moreover,

there is a progressive destruction of ferments along the alimentary canal, the gastric juice destroys the activity of the saliva, and the pancreatic juice destroys the gastric.

Pathology of external secretions.—In disease secretions are frequently affected, both in respect of the quantity of water eliminated, and of the nature and quantity of the essential constituents. The *salivary and buccal secretions* may be totally arrested, as in cases of xerostomia where great difficulty in swallowing arises from the mere lack of lubrication. A converse condition of excessive or of continuous secretion arises when there is some reflex irritation, especially of the mouth and tongue; as in cases of epithelioma of the tongue. Continuous dribbling of saliva occurs in bulbar palsy, and this condition may be due to the difficulty in swallowing; yet in some cases the quantity of saliva seems excessive, and suggests a "paralytic secretion." Very rarely, and without the presence of any gross organic disease, copious and persistent salivation occurs, giving rise to a condition exactly opposite to that seen in xerostomia. The nature of these cases is obscure; they are not due to the taking of such drugs as the iodides, and they too seem to resemble the "paralytic secretion" of the physiologist.

The secretion of sweat is also profoundly affected in various diseased conditions; and in febrile disorders, for instance, the secretion may be increased, diminished, or arrested. When increased, the increase is by no means always proportionate to the height of the fever. This is more especially true of phthisis, where very copious sweating may occur with comparatively little fever, and with a pale, anæmic skin. A direct contrast to this is seen in the dry and burning skin of pneumonia. The mechanism of the sweats in fever is not clear, but it is probable that they are dependent upon the excitation of the sweat centres and glands by abnormal substances present in the blood-stream, as well as upon the mere increased temperature. Heat causes sweating by its action as a stimulus to the nerve-centres. Copious perspiration is seen also as a result of pain, especially of reflex origin, as in biliary and renal colic. A peculiar instance of reflex excitation of sweating is that occasionally seen involving the face and neck, and occurring during mastication. Sweating confined to small areas often occurs as a result of nervous lesions, especially as a result of pressure on such a nerve as the cervical sympathetic, or from disease involving the central gray matter of the spinal cord. In syringomyelia very copious sweating is often observed, and the transition from the area of copiously sweating skin to the adjacent dry area is often sudden. The sweating often coincides in its distribution with the analgesia so often seen in this disease. Conversely the activity of the sweat-glands may be diminished in disease, as illustrated by the dry skin of diabetes and renal disease.

The varying degrees of activity of the sweat-glands have a powerful effect in the regulation of the body temperature, since the skin and its sweat-glands form one of the channels by which variations in the loss of heat are regulated.

The urinary secretion is considered in detail elsewhere [*vide* art. on "The Urine" in the following volume]. Variations in the amount of urine excreted depend largely upon the amount of water lost by such other channels as the skin, the lungs, and the alimentary canal; next to this the rate of flow through the renal vessels is the most important factor in determining the quantity of water excreted by the kidneys.

In diabetes insipidus, on the one hand, and in the complete suppression seen after reflex irritation of various organs in disease, on the other, the nervous system must play an important part; but whether it acts directly on the renal cells, or only on the blood-vessels, is not definitely known. Relatively little is known of the effect of disease on the quantity of the digestive juices. The gastric juice is held to be diminished in amount in many diseases, and especially in organic diseases of the stomach; but much more is known of the changes in its composition. In gastritis, and in carcinoma ventriculi, the quantity of pepsin, and also the amount of hydrochloric acid, are greatly diminished, and may sometimes be completely absent. In some so-called functional derangements of the stomach, and in gastric ulcer, the quantity of hydrochloric acid is said to be increased.

In many conditions, but more especially as a result of anxiety and nervousness, copious watery stools are passed; but it is probable that this discharge is dependent rather upon an undue hurry of the contents of the intestine than upon any actual increase in the amount of enteric juices.

Calculi.—In glands provided with ducts, morbid processes often lead to the formation of calculi, which consist usually of some of the more abundant constituents of the secretion. Occasionally the calculus-forming material is one, such as indigo, that normally is present in traces only; in some cases, indeed, as in that of cystine calculi, it is doubtful whether the substance be a normal constituent of the secretion at all. Purely inorganic stones are rare, but inorganic stones formed by accretion about an organic nucleus are not uncommon.

The mechanism of calculus formation is sometimes obscure, but usually it is related to one or more of the following conditions:—

(i.) An excess of the petrifying constituent in the secretion. This mechanism is perhaps not so frequent a cause as was formerly supposed; but in many cases, no doubt, it holds an important place in the causation. Deposits of calcium oxalate occur not infrequently in the urine as a result of the ingestion of material containing considerable quantities of oxalate. Uric acid concretions in the kidney depend sometimes, perhaps, upon an excess of the acid in the urine. Usually a mere excess of a particular ingredient normally excreted in a soluble form is not of itself sufficient to determine the formation of stone. Uric acid concretions, indeed, may be found in the urine when the percentage of this acid is small and the urine dilute; on the other hand, large quantities of the acid may be excreted without the formation of stone. Gall-stones are sometimes the result of the presence of an excessive amount of cholesterin in the secretion.

(ii.) An alteration in the composition of the secretion so that the relative proportions of the various constituents are altered, and thus the chemical interactions dependent upon "mass action" are also altered.

This is a most important cause of stones, and the formation of uric acid gravel often illustrates it. The uric acid in the urine is in the form of a soluble quadriurate, and for this salt to be formed it is necessary that a certain percentage of inorganic salts should be present. If this percentage be diminished uric acid may be precipitated as such, and a stone may be formed without the presence of any excess of uric acid in the secretion at any time. In the case of the biliary secretion an increased amount of calcium salts in the bile may lead to the formation of a biliary calculus, calcium forming an insoluble compound with the bile pigments.

(iii.) The size and shape of the crystals of certain salts are altered by the presence of colloidal substances in the secretion.

Oxalates and other crystalline substances can be made to crystallise out from their solutions by the addition of some colloid matter such as albumin. The substance in solution not only crystallises out, but the crystals are frequently very large, and in this way may act as a nucleus for subsequent crystalline accretion. The exudation of blood or of some albuminous material into the renal pelvis may perhaps determine the formation of a stone in this way, and may serve to explain the occurrence of stones in one kidney only; although the calculus-forming material is probably present to an equal amount in the blood distributed to both kidneys.

Crystals of uric acid are affected profoundly in their size and shape by certain pigments in the urine, and thus variations in the amount of urinary pigment may modify the form in which the uric acid is excreted.

(iv.) It is possible that colloid material, such as nucleo-albumins, may act as a nucleus for the formation of stones. This action is distinct from the preceding, in that it is simply mechanical, whereas the essence of that described in the third section lies in the fact that the crystalline form is altered, at any rate as regards the size of the individual crystals. Gall-stones are sometimes formed long after an attack of catarrhal jaundice, and apparently as a sequel of it. It is possible that in these cases a cholangitis has extended to the gall-bladder, and that the exudation from the mucous membrane of the gall-bladder has determined the precipitation and crystallisation of the calculus-forming substance. In rare instances foreign bodies have been found as the nuclei of stones, but even in these cases it is possible that the foreign bodies produce the effects by means of inflammatory changes in the mucous membrane.

Diseases of glands resulting from partial or complete obstruction of the ducts are common. The duct of a gland is liable to become obstructed as a result of a mere alteration in the consistency of the secretion; from the presence of calculi; from the results of inflammatory changes in the walls of the duct, or from the pressure produced by growths in the duct or in adjoining structures. When the duct of a gland is obstructed the secretion does not cease at first; the usual effect is that the gland-ducts,

and later the gland itself, become distended with a more or less abnormal secretion. Some of the constituents of the secretion pass into the circulation, either directly through the vessels of the gland or indirectly through the lymphatics. In many cases of long-continued obstruction the effects are complicated by the occurrence of inflammation of the walls of the duct. The ducts become greatly distended with more or less stagnant secretion, and under the circumstances an ascending microbic infection is liable to occur. This is especially seen in the case of obstruction of the bile-ducts.

In the salivary glands, pancreas, and kidneys the secretion goes on at a sufficient pressure to distend these organs forcibly to a considerable size. In the liver the bile is secreted normally under a low pressure; thus jaundice results even when the obstruction to the biliary flow is slight, the stagnant bile passing into the lymphatics and veins of the liver.

Toxic substances.—Secretions in disease frequently contain abnormal and more or less toxic substances, and thus act as the means of their riddance. In uræmia, the bile, the gastric secretion, and the sweat contain appreciable quantities of urea and other extractive bodies. In cases of hæmoglobinuria, due to the presence of free hæmoglobin in the blood plasma, the hæmoglobin is excreted not only in the urine but in the bile also.

These abnormal bodies are not taken up indiscriminately by all glands. Bile pigments, in cases of jaundice, appear readily in the urine; but even in cases of complete and persistent obstructive jaundice bile salts appear in the urine in but small quantities. The different secretions exert a more or less selective action upon the abnormal constituents removed by them from the blood-stream.

Microbic poisons are frequently removed from the body in secretions, more especially in the urine; if microbes are present in the general blood-stream the urine may contain them also. It is probable that many pathological processes, such as gastritis or nephritis, are dependent upon the removal by the stomach or kidneys of toxins and microbes from the general circulation. However, all disease poisons present in the blood are not necessarily present in glandular secretions; thus the virus of syphilis, although present in the blood, is not necessarily present in the proper secretions of the syphilitic. The saliva as secreted from the salivary glands of syphilitic persons is not infective, although the saliva in the mouth may be infective as the result of contamination with some exudation from a syphilitic lesion in the mouth. It is denied that the virus of rabies, although present in the nervous system and excreted by the saliva, is contained in the urine and bile.

INTERNAL SECRETIONS.—In the case of internal secretions the gland elaborates some substance or substances that are required to maintain the metabolic activity either of the tissues generally or of some particular tissue. The existence of internal secretions is most easily established in the case of glands without ducts, such as the thyroid and suprarenals;

the difficulty is greater in cases like the liver, pancreas, and kidneys: yet even in these cases there is some evidence of the existence of internal secretions. The evidence on which the existence of internal secretions is based in the case of ductless glands, such as the thyroid, is as follows:—The complete removal of the gland is followed by death, and this event cannot be attributed to any lesion of the nervous system produced at the operation. Death is dependent solely on the removal of the gland: because, first, if a fragment of the gland of a certain size be left, death does not ensue; secondly, if a portion of the gland be successfully grafted in the subcutaneous tissue, and if the graft acquire proper vascular connection, the entire normal gland may be removed without fatal consequences; thirdly, in some cases of the removal of the gland survival is prolonged by the administration of an extract prepared from the gland substance.

Thyroid.—The complete removal of all thyroidal tissue is fatal in all animals hitherto experimented on, and in man. The removal of the thyroid body, however, is not necessarily fatal, since thyroidal tissue may be present in certain bodies known as accessory thyroids and parathyroids. Accessory thyroids have the same structure as the thyroid itself, but the minute anatomy of parathyroidal tissue is somewhat different in that the cells which enter into this tissue are not arranged to form the lining membrane of a colloid-containing vesicle; in the parathyroids the cells are arranged in columns resembling those of a compound tubular gland, there are no large alveoli and no colloid. The parathyroids are sometimes—as in the rabbit—at some little distance from the thyroid itself, sometimes—as in the dog—embedded in the upper extremity of each lobe of the thyroid, and sometimes—as in the monkey—embedded in the substance of the thyroid itself.

Parathyroidal and thyroidal tissue do not play an equivalent part in preventing the development of the symptoms which follow thyroidectomy. In the rabbit the thyroid itself can be removed completely without permanent ill effects, as the parathyroids are left untouched by any such operation. Whether in the dog complete removal of one lobe together with the greater part of the opposite lobe lead to survival or death depends upon whether one parathyroid is left or not; that is, if one lobe be entirely removed life may go on in the dog after excision of the bulk of the opposite lobe, provided the upper extremity containing the parathyroid be left: if, however, this upper extremity be removed a much larger fragment of the thyroid (amounting from a half to two-thirds of the lobe) must be left. On the other hand, the complete removal of the parathyroids only is followed by no obvious ill effects. Thyroid grafts prolong life after complete thyroidectomy; but it is remarkable that the injection of thyroid extract does not, in the monkey at any rate, prevent with certainty the development of the specific symptoms seen after this operation. In dogs, also, thyroid-feeding after thyroidectomy only secures a brief prolongation of life; in but a few cases does it prevent an early death. Complete removal of all thyroidal tissue is followed in the acuter cases by the rapid appearance of a series of symptoms of which

tremor, tetany, clonic spasms, and dyspnœa are the chief; in other cases, where survival is more prolonged, a curious swelling of the parotid and other regions ensues, the hair becomes brittle, and more or less complete alopecia results. Clonic spasms are most marked in the carnivora, and in them the operation is followed by death, usually within a few days. The clonic spasms are accompanied by dyspnœa with paroxysmal exacerbations. In the dog, albuminuria in considerable amount occurs also after the thyroidectomy. The group of symptoms produced suggests in many ways a toxæmia, and some authors have held that the blood is thus tainted; others, however, have failed to reproduce the symptom group by the injection into normal animals of blood obtained from animals after thyroidectomy. In man removal of the thyroid is followed by a condition closely allied to myxœdema, if not identical with it; but not infrequently tetany also has been so produced. The experimental myxœdema, seen in animals surviving from the acute and initial symptoms, apparently depends upon a disordered metabolism, and this condition in the human subject can be arrested and held in check by the administration of thyroid extract; hence it is clear that whatever other functions the thyroid may possess it supplies at any rate a substance necessary for normal tissue metabolism.

The thyroidal tissue left after partial thyroidectomy undergoes a series of changes. The fragment increases considerably in size, but the structure of the normal thyroid is not accurately reproduced. The epithelium lining the alveoli proliferates, so that in places the alveoli are no longer lined by a single layer of cells; on the other hand, there is no formation of colloid, so that the structure of the hypertrophied thyroid resembles the structure of the normal parathyroids.

The parathyroids after partial thyroidectomy undergo slight enlargement, but there is no abnormality in their structure and no formation of colloid.

The thyroid body contains a large quantity of a complex proteid substance that contains, in addition to the usual constituents of proteids, a considerable proportion of iodine and some phosphorus. This proteid body can be split up by a variety of methods, amongst others by digestion yielding on the one hand albumoses, and on the other a non-proteid substance containing a considerable percentage of iodine—the so-called thyroiodin. It is probable that this complex proteid substance, yielding this "thyroiodin" as one of its decomposition products, is the main ingredient of the colloid matter present in the thyroid. Although the non-proteid moiety of this complex substance contains a large quantity of iodine, a smaller proportion of iodine is also contained in the proteid portion of the original colloid substance. The name "thyroiodin" was first applied to a non-proteid substance, containing iodine, obtained by treating thyroids with strong acids; but this substance is probably identical with that described above as a decomposition product of the colloid material. Both the proteid moiety and the non-proteid moiety of the colloid substance are described as "active," that is, they both produce the physiological and therapeutical properties of thyroid extract;

but the non-proteid substance containing the larger percentage of iodine is by far the more active of the two.

Formerly the colloid substance was looked upon more or less as an excretion; now, however, it is regarded as an internal secretion, and in confirmation of this the colloid has been found in the lymphatics of the gland, suggesting that it is being carried away to supply the requirements of the body, and perhaps more especially those of the nervous system. If the colloid substance containing this thyroiodin is the essential and specific substance elaborated by the thyroid, it is remarkable that life can be maintained, in the carnivora at any rate, when the parathyroids, or even one parathyroid is left; for these bodies contain no colloid.

The Suprarenals.—Removal of one suprarenal in animals, according to most observers, is not followed by any serious or permanent derangement of health; but Tizzoni found that death followed unilateral extirpation not infrequently, and that the operation was followed by a series of changes in the central and peripheral nervous system [*vide* art. "Addison's Disease," vol. iv.] There is far less agreement as to the results of bilateral extirpation; some observers have found that death ensues within a few hours, others after a period of varying duration of cachexia and marasmus; others again state that complete bilateral extirpation is possible without either death or illness. It is possible that these differences, extreme as they are, may be due partly to the operative procedures adopted, and partly to the presence of accessory suprarenals. Those authors who have alleged that bilateral removal of these glands is followed by death from marasmus have also described hypertrophy of the remaining gland after unilateral extirpation; and further, that successful grafting of an adrenal will prevent the fatal effects that usually follow bilateral extirpation: but it is said that, for the graft to be successful, it is essential that it should be derived from an animal of the same species.

Death after bilateral extirpation of the suprarenals has usually been preceded by a period of marasmus, often extreme; but in a few cases some observers have noted the appearance of pigment in situations normally more or less free from pigment. As in the case of the thyroid, so in the case of the suprarenals, it may be asked whether the fatal event be due to the damage done to the sympathetic nerves during the operation, or whether it be directly and solely dependent on the removal of the gland; and in the latter case whether death be due to the accumulation of some toxic material, or to the suspension of an internal secretion.

It is asserted that after complete removal of the suprarenals a condition of partial palsy supervenes, which depends upon the presence in the blood of a curara-like poison; and that if the blood of an animal in this condition be injected into a normal animal, it produces in the latter a similar train of symptoms. On the other hand, there is clear and definite evidence that the suprarenals contain, and probably secrete into the suprarenal vein, a substance capable of causing profound effects on muscular tissue, and more especially on the muscular tissue of the arteries. Suprarenal extract, when injected into the circulation, produces a sudden and

great rise in arterial pressure, which rise depends upon contraction of the peripheral arterioles. This contraction of the muscular coat of the arterioles is brought about by a direct local action of this substance on the muscular coat, and not by any action on the vaso-motor centre. This active substance, whose chemical composition is very obscure, is contained most abundantly, if not exclusively, in the medullary portion of the gland, and its potency is such that an extract representing $\frac{1}{1,000,000}$ of the weight of the dried gland, when injected intravenously, will produce a very notable effect on the general blood-pressure. It is at least doubtful whether the phenomena alleged to follow extirpation of the suprarenals can be correlated with any failure of the circulation dependent upon the absence from the blood-stream of this tonus-producing substance. Whatever doubt may remain as to the functions of the suprarenals, there can be no doubt as to the existence of an internal secretion; although perhaps it is not certain that the internal secretory activity of these glands is their sole function.

Pancreas.—The pancreas affords another instance of a gland where there is very definite evidence in favour of the existence of an internal secretion. In dogs the removal of the entire pancreas is followed by a condition of glycosuria, accompanied by great wasting and thirst, and ending soon in death—a condition far more entitled to the name of experimental diabetes than the more or less transitory glycosuria producible by that puncture of the bulb known as the "diabetic puncture." This fatal pancreatic glycosuria is not seen after ligature of the pancreatic duct, nor after removal of the bulk of the pancreas, provided only that a small fragment of the gland be left; but if this fragment be excised by a second operation, the fatal disorder ensues. For these reasons the glycosuria cannot be due to mutilation of the abdominal nervous structures, but must be regarded as due to the removal of the pancreas itself.

The fatal effects following the removal of the pancreas can be prevented by the successful grafting of a portion of the pancreas in the subcutaneous tissue, just as successful thyroid grafting will prevent the fatal effects of complete thyroidectomy. Very exceptionally dogs have survived the operation of excision of the pancreas, and in such rare cases no glycosuria has ensued [*vide* art. "Diabetes," p. 208].

The existence of experimental pancreatic glycosuria is unquestionable; the mechanism of its production is obscure. The hypothesis of the formation by the pancreas of a glycolytic ferment secreted not into the duct but into the lymphatics and blood-vessels, which therein assists in the decomposition of the sugar of the blood, although plausible, is not clearly established.

Liver.—The sugar-forming function of the liver, that is, the conversion of the glycogen of the liver into sugar to be distributed to the body at large, is really an instance of an internal secretion. Some authorities, however, deny that the liver is a sugar-forming organ, and regard the glycogen as a precursor of fat; the great bulk of physiological opinion, however, is in favour of the former view.

Kidneys.—Experiments performed by myself have demonstrated that

after removal of the greater part of both kidneys the excretory functions of the organ continue not only undiminished, but indeed that the amounts of urine and urea excreted may actually be increased in amount; and further, that this disordered metabolism may lead to great wasting and marasmus, followed by death. This disordered nutrition is entirely dependent upon the amount of kidney removed; hence it is possible that the phenomena are due to the arrest of a normal internal secretion. This result cannot be considered proved until the progress of the disordered nutrition can be arrested by the injection of kidney extract, or by the successful grafting of kidney substance. This, however, has not as yet been done successfully.

Sexual glands.—The testes and ovaries produce, as is well known, a profound effect in the economy; since not only the general nutrition, but also the whole or the main characteristics of the organism, may be altered by the removal or degeneration of these glands. It is not known whether these glands produce their effects by the chemical stimulus of an internal secretion or by a reflex action on the nervous system. The removal of the organs, in the human subject at any rate, is occasionally followed by remote general effects which it seems difficult to attribute to the arrest of an internal secretion. Phenomena of this nature are the mental disturbance seen sometimes after the removal of the uterus, and perhaps the atrophy of the prostate that is said to follow removal of the testes. It is possible that the removal of glands, like the removal of organs, may produce remote effects that cannot be attributed to the presence of an internal secretion; at the same time the remarkable discovery of the substance elaborated by the suprarenal bodies shows what totally unexpected and profound effects an internal secretion may bring about.

The existence of an internal secretion in other glands is improbable. The removal of the salivary glands is not followed by any obvious effects. The spleen can be removed completely without ill results, although in certain cases the resistance of the animal to microbic infection is diminished. The complete removal of the pituitary body does not produce any obvious ill effects, at any rate within a short interval after the operation. It has been asserted, however, that the pituitary body undergoes enlargement after thyroidectomy, and thus some observers have considered that this body is accessory to the thyroid. The experiments of Schäfer and Oliver, however, indicate that the physiological action of the extracts of the thyroid and pituitary bodies may be antagonistic, not complementary.

Pathology of internal secretions.—The proof of the existence of internal secretions has given an impetus to pathology, and has led to the hypothesis that disease may be the result of one or more of the following variations:—

I. The arrest of an internal secretion.

II. The excessive formation of an internal secretion.

III. The abnormal composition of an internal secretion.

In many cases these views of the pathology of diseased conditions are

little more than surmises, in others they are more securely established; this is especially the case with reference to the diseases of the thyroid body. Such conditions as myxœdema and sporadic cretinism are held to be due to the more or less complete arrest of the internal secretion of the thyroid, and on the other hand exophthalmic goitre is thought by some pathologists to be due either to a hypersecretion or to an abnormal secretion of the thyroid. The view of the pathology of myxœdema is more securely established than that of exophthalmic goitre. Not only can a condition allied to myxœdema be produced by the removal of the thyroid in man and animals, but also the effects of myxœdema in the human subject can be removed by the administration of thyroid extract, and perhaps by thyroiodin. In the case of exophthalmic goitre the matter is not so simple; the phenomena produced by the injection of thyroid extract are not quite similar to those seen in exophthalmic goitre. That the exophthalmos present in this disease may be unilateral has been urged as an objection to the view that it is produced by a poison circulating in the blood. However, metallic poisons, like arsenic and lead, will sometimes produce unilateral lesions of the nervous system, so that this objection is not a very powerful one. A more serious objection is that the general symptoms of the disease do not necessarily vary step by step either with the magnitude or with the rate of development of the goitre. Enlargement of the thyroid, very similar to that seen in Graves' disease, may exist without any of the symptoms of the latter malady. The changes in the thyroid body in Graves' disease are apparently very similar to those seen in animals after the removal of a portion of the thyroid. The enlargement is of such a character as to reproduce the structure of the normal parathyroid rather than that of the normal thyroid. The enlarged thyroid consists of alveoli lined with actively proliferating cells, and these alveoli contain no colloid. The fact that the thyroid gland in this disease has a microscopic structure analogous to that seen experimentally when a fragment of the gland is left and undergoes enlargement, has suggested to some observers that the enlargement of the thyroid in exophthalmic goitre is not primary, but secondary, and arises in response to some need of the economy. It is possible, however, that when the thyroid enlarges, whether primarily or secondarily, the form and structure of the enlargement might be similar. The clinical observation that exophthalmic goitre is sometimes followed by myxœdema certainly suggests that the former disease is dependent on the thyroidal hypersecretion.

On turning to the other glands we find that the evidence available at the present time to prove that certain diseases are dependent upon disorder of the function of internal secretion is by no means so strong.

In Addison's disease the interesting observation has been made that the remains of the suprarenals found in this disease contain no active suprarenal extract. At present, however, it is not possible to give a complete explanation of the phenomena seen in Addison's disease on the hypothesis that all of these result from the arrest of the internal secretion. Yet it is possible that the weakness of the circulation may be due to the

absence from the blood-stream of the powerful tonus-producing substance elaborated by the normal suprarenal. Although some success has been achieved in treating cases of Addison's disease with suprarenal extract, no such striking results have as yet been obtained as those seen in the treatment of myxœdema with thyroid extract.

In the case of the pancreas and diabetes the present state of our knowledge is also unsatisfactory. It is certain that diseases of the pancreas may be accompanied by diabetes, but the association is not an invariable one. Cancer of the pancreas may exist with or without diabetes, and many cases of diabetes are seen with no gross lesion of this gland, although cirrhosis has been frequently discovered on microscopic examination.

Certain pathologists have thought that lesions of the pancreas are associated with a particular variety of diabetes running an acute course. Lesions of the pancreas cannot, however, be regarded as causing all varieties of diabetes, and it is probable rather that glycosuria is a symptom that can be produced by various lesions in different organs. Treatment of diabetic patients with pancreatic extract has been followed by very little success, if any.

In the case of diseases of the kidneys, the question of the existence of an internal secretion will be discussed in the section on pathology of the kidneys in the next volume.

JOHN ROSE BRADFORD.

REFERENCES

1. BAUMANN. "Jod in Thierkörper," *Zeitschrift f. phys. Chemie*, 1896.—2. BRADFORD. "Gland Nerves," *Journal of Physiology*, 1888.—3. *Idem.* "Influence of Kidney on Metabolism," *Proc. Roy. Soc.* 1892.—4. EDMUNDS. "Pathology of Graves' Disease," *Path. Soc. Trans.* 1895-96; *Proc. Roy. Soc.* 1896, and *Journal of Pathology*, 1896.—5. GLEY. *Comptes Rendus Soc. de Biol.* 1891-92; *Archives de Physiologie*, 1892-93-94.—6. HALSTED. "Experimental Study of Thyroid," *Johns Hopkins Reports*, 1896.—7. HEIDENHAIN. "Ueber secretorische und trophische Drusen nerven," *Pflüger's Archiv*, 1878.—8. HORSLEY. "Functions of the Thyroid," *Proc. Roy. Soc.* 1885-86, and *Virchow's Festschrift*, 1891.—9. HUTCHISON. *Journal of Physiology*, 1896.—10. LANGLEY. "Salivary Secretion," *Journal of Physiology*, vols. i. ii. vi. ix. x.—11. LÉPINE. "Le pouvoir glycolytique du sang," *Comptes Rendus*, 1890.—12. MINKOWSKI and V. MERING. *Archiv f. exp. Path. und Pharm.* 1890.—13. MUNK. "Untersuchungen über die Schilddrüse," *Sitzungsberichte der k. Akad.* Berlin, 1887.—14. MURRAY. "Treatment of Myxœdema," *British Med. Journ.* 1891, and *Lancet*, 1893.—15. SCHÄFER and OLIVER. "The Physiological Effects of Extract of the Suprarenal Bodies," *Journal of Physiology*, 1895.

J. R. B.

SHOCK AND COLLAPSE

THE name shock is applied to the state of prostration which may affect those whose bodies have been severely injured, or whose minds have been shaken by intense emotion. A state of prostration very similar to the above, but affecting those suffering from disease, from the more

remote effects of injury, or from the action of certain poisons, is known as collapse.

Between shock and collapse there is considerable resemblance: both are states of prostration, and in both the circulation of the blood is impaired. The pathology of these states having been imperfectly understood, the distinction between them has rested hitherto upon differences in the conditions under which they occur; but in the course of the following pages evidence will be brought forward to establish a pathological distinction between them. It will be shown that two kinds of prostration occur, somewhat similar, indeed, in their outward manifestations, and both mainly dependent upon failure of the circulation, but differing essentially in the pathological changes which cause the circulation to fail.

Some other examples of prostration remain, the pathology of which is still obscure: I refer particularly to those which are caused by the action of certain poisons. Of these, some may enter the body from without; others may be formed within the body. When the mode of action of these substances is fully understood, it is possible that the prostrations which they respectively produce may in most cases be recognised as belonging to the one or the other of the varieties which I have indicated.

The causes of shock.—Shock is closely allied to syncope, but differs from it in that the prostration is more prolonged, and that consciousness though blunted is not usually abolished. It is often complicated by hæmorrhage or by direct injury to the brain. With these incidents this article is not concerned. Strong emotions alone, such as fear, anger, or grief, may cause shock; but more usually they combine with bodily injury to produce this state; thus Sir William MacCormac states that the wounded soldiers of a defeated army are liable to suffer more severely from shock than the equally severely injured men of the victorious side. The temperament of the sufferer is doubtless an important factor in the matter. As an example of the influence of the emotions on shock I may mention that when on one occasion I removed the tonsils from a boy of nervous temperament, this little operation was followed by so much prostration that the patient remained semi-conscious and almost pulseless for several hours. But while an emotional disturbance alone may cause shock, yet this state usually follows severe bodily injury; such as the crushing of a limb, an extensive burn, a severe operation, the constriction of a loop of intestine, or the passage of a renal or biliary calculus. Slight injuries, however, may be followed by shock, especially when they affect important organs; and a blow upon the epigastrium, even of no great force, may cause a considerable degree of prostration. Pain is probably a factor of some importance; for an injury to a sensitive part, such as the testicle or finger, is followed by a greater degree of shock than is an injury of equal severity to some less sensitive part. But that injury, quite apart from the sensations or emotions to which it may give rise, is capable of producing shock, is

shown by the fact that anæsthetics have not abolished shock after surgical operations; although they have very considerably reduced both the severity and the frequency of its occurrence. It will be observed that *the causes of shock are such as may be expected to produce an effect upon the central nervous system, either directly, or by means of afferent nervous impulses.*

The causes of collapse.—Collapse occurs after abdominal operations, and in the course of those diseases, such as cholera, of which diarrhœa and vomiting are the prominent features. Now persistent diarrhœa and vomiting are the cause of considerable loss of fluid from the body; and, as will be presently shown, there is, after abdominal operations and presumably also in peritonitis, a considerable escape of fluid from the blood into the injured tissues. In none of these conditions is there any reason to think that the central nervous system is greatly affected.

The action of certain poisons upon the body causes a condition of prostration, which is usually called collapse. Some of these, no doubt, act locally upon the walls of the alimentary canal, and, by causing diarrhœa and vomiting, lead to thickening of the blood; but many of them cause prostration without having induced either of these symptoms. Of these latter there is reason to think that some act directly on the nerve-centres, others on the heart and blood-vessels; others, again, may possibly produce changes in the blood itself. The kind of prostration which is caused by the poisons which affect the circulation, by acting either directly upon the heart and vessels, or indirectly through the medium of the nervous system, is in its pathology so closely related to that which is produced by mechanical injury that it would seem better to consider it as a variety of shock rather than of collapse.

Symptoms.—Shock usually follows quickly the injury or emotional disturbance which is the cause of it; but it may be delayed for a short time during which the stimulus of excitement keeps up the activity of the nervous centres. Collapse, on the other hand, comes on insidiously, several hours after an injury or operation, or during the course of disease. In other respects the clinical features of these two conditions are so similar that for convenience they may be described together. The similarity is sometimes so great that a case of intestinal strangulation has been mistaken for one of cholera sicca (6). On the other hand, patients suffering from the collapse of cholera often do not present the extreme prostration of those who have been severely injured. They may have no sense of faintness, and be able to sit up, or even to walk about, when no pulse can be felt at the wrist (6).

In extreme cases both of collapse and shock the patient has the appearance of profound prostration; he lies perfectly quiet, and gives no heed to that which is passing around him.[1] Consciousness, though

[1] The condition of "prostration with excitement," described by Travers as a variety of shock, is considered by Mr. Watson Cheyne (18) to be due in part to the action of bacterial products absorbed from a wound into the blood. It is better, therefore, to regard it rather as a complication than as a variety of collapse.

seldom completely absent, is blunted even in mild cases; and it is with difficulty that we elicit from the sufferer answers to questions; yet if we succeed in so doing their import is usually rational and signifies that the intellectual faculties are clear.

The face is pale, the lips are livid, and the features are thin and drawn. There is indeed in severe cases, more especially in the collapse of children suffering from continued diarrhœa, an appearance of emaciation out of all proportion to the duration of the illness. The eyes lie sunken in dark hollow orbits; the nose is thin, and through the wrinkled skin the contours of the facial bones appear. The hands and other parts of the body show changes of the same kind. The surface of the body is cold and moist; the internal temperature is usually subnormal, but in cholera and other varieties of diarrhœa it may be raised. To obtain evidence of this rise the thermometer must be placed in the rectum; for, even when the internal temperature is considerably raised, the mouth may be cold, and the breath fail to communicate a sense of warmth to the hand of the observer. The pulse is irregular and usually rapid, and becomes more and more feeble, as the general condition gets worse, until it can no longer be felt at the wrist. The breathing is accelerated and irregular, and the patient complains of want of air. The secretion of urine is diminished, or may be arrested, as indeed all the secretions probably are, except those of the skin and of the tissues directly affected by disease or injury; such, for example, as the peritoneum after abdominal operations or during peritonitis, and the mucous membrane of the intestines in cases of diarrhœa. The patient suffers severely from thirst, and the mouth and tongue are dry. The vocal cords also are probably dry, for the voice is not only feeble but altered in quality and has a harsh tone. The reflexes are slow and the urine is sometimes retained, even if it be secreted.

Pathology of shock.—The well-known effect of stimulating the central end of the depressor nerve of the rabbit, or of striking the abdomen of the frog (8), leaves no doubt that the circulation can be profoundly modified by impulses which reach the vaso-motor centre. Such procedures, by dilating the arterioles and so lessening the peripheral resistance to the circulation, cause a fall of arterial blood-pressure.[1] It would seem, in the latter case at least, that the walls of the veins also are dilated; for the more rapid flow of blood through the arterioles does not cause an increase of the intake of the heart; on the contrary, this is diminished in a marked manner, particularly if the animal be held with its head upwards.

From these and other similar experiments it has been inferred that

[1] The effect of striking the abdomen of the frog is not only reflex vaso-dilatation, but also a stoppage of the heart in diastole for a few seconds. Stimulation of the central end of the vagus or of the mesenteric nerves of the mammal has the same effect. This effect, however, is transient, and although probably the cause of syncope or even of the sudden death which sometimes follows a blow on the abdomen, it cannot be called upon to explain that protracted failure of circulation which constitutes shock.

when a portion of the body has been severely injured, or the brain disturbed by intense emotion, afferent impulses arise from the part affected, reach the vaso-motor centre in the medulla, and by means of impulses thence reflected cause the arterioles of the body generally, and of the splanchnic area in particular, to dilate; thus the arterial blood-pressure is lowered. In consequence of this process so large a quantity of blood accumulates in the abdominal veins that the volume of blood passing through the rest of the body is seriously reduced.[1]

The anæmia of the superficial parts of the body which is produced in this way is probably accompanied by a diminution in the quantity of lymph in these parts; for Dr. Lazarus Barlow has shown that the withdrawal of blood from one part of the body to another is accompanied by a rise in the specific gravity of the tissues which become anæmic. This diminution of lymph, if it occur in shock, affords a satisfactory explanation of the pinched and shrunken appearance of the patient, to which the name *Facies Hippocratica* is usually applied. On the other hand, the hyperæmia of the abdominal vessels does not lead to a corresponding outflow of lymph from them; for, according to Prof. Sherrington and Dr. Copeman (15), it is followed by a fall in the specific gravity of the blood; at any rate when it is caused by division of the spinal cord. I am not aware of any direct observations on the changes, if any, in the specific gravity of the blood which occur in cases of uncomplicated shock in man; but from what has been said, it seems probable that it will be found lower than the normal—in this respect presenting a marked contrast to what is found in the prostration of cholera.

But dilatation of the abdominal vessels does not afford a complete explanation of the phenomena of shock, for they differ in many important respects from those of mere hæmorrhage. Years ago, John Hunter (11) observed in a person suffering from syncope that the venous blood was of a bright red colour; and he argued that this could not be due to a more rapid flow of blood through the capillaries, because it occurred when the circulation was very languid. More recently M. Roger, following Brown-Séquard, has observed this phenomenon in animals in which shock has been experimentally induced, and attributes it to an arrest of the exchanges which normally take place between the blood and the tissues, as a consequence of which the oxygen in the blood passes through the capillaries and is

[1] It is true that the usual effect of stimulating the central end of the divided sciatic nerve, or of any nerve containing afferent fibres, is a rise of arterial pressure; but if the animal be under the influence of chloral, a fall of pressure results instead. In the words of Professor Foster, "The condition of the central nervous system seems to determine whether the effect of afferent impulses is one leading to an augmentation of vaso-constrictor impulses and so to a rise, or one leading to a diminution of vaso-constrictor impulses and so to a fall of blood-pressure" (7). When a rise of pressure occurs on stimulating a sensory nerve, it is but transient, and often disappears before the stimulus has ceased to act. It may be that the continued excitation of sensory nerves, which results from injury to some peripheral part, exhausts the vaso-motor centre, and leads ultimately to a fall of blood-pressure. But of this there is no direct evidence.

not taken up by the tissues. He finds, moreover, that strychnine injected into the veins of a frog does not produce any effect if the animal be in a state of shock caused by crushing its head; but it may do so at a later period when shock is passing away. It has long been known to surgeons that patients in this state often fail to respond to the administration of opium and alcohol, but may afterwards exhibit symptoms of poisoning if the drugs have been given in large quantities. The arrest of their action in shock has usually been attributed to want of absorption from the stomach. According to Roger it is due to arrest of the vascular exchanges; and this change leads also to a diminution of the production of heat, and is the cause of the fall of temperature which is found in shock. For this observer the arrest of the vascular exchanges is the most important change which occurs in shock; it is the only one of a series of inhibitory effects caused by violent excitation of the nervous system which is constant and indispensable.

The truth of these conclusions has been disputed by M. Contejean, who maintains that in Roger's experiments the circulation of the blood was impaired, and that the strychnine was further prevented from reaching the spinal cord in consequence of the blood-vessels of that organ having been interfered with by the means taken to cause shock; and that if the head of the frog be crushed in such a way as to leave the blood-supply of the cord intact, then strychnine produces its usual effects. But he goes on to say that shock induced after the administration of strychnine can arrest tetanic spasms. Of this apparent contradiction he gives no explanation. M. Roger has replied by showing that when shock is thus induced in such a manner as not to interfere with the spinal cord—namely, by the discharge of a Leyden jar applied to the lumbar region—the action of strychnine is nevertheless suspended for a time; moreover, that veratrine given under similar conditions does not prolong the period of contraction of voluntary muscles. He therefore adheres to his belief that in shock there is an arrest of the vascular exchanges. The experiments of neither of these authors are in my opinion conclusive.

At the present time the theory of shock which finds most common acceptance is that this condition is caused by nervous impulses of great intensity, which may arise either in the cerebrum itself or in some distant part. These depress the activity not only of the vaso-motor centre but of the whole nervous system also. In other words, it is believed that the symptoms are not merely the consequence of failure of the circulation, but are caused by a depression of cerebral activity, of which the failure of circulation is but one of the effects. In short, to use the words of Mr. Mansell Moullin, "Shock is an example of reflex paralysis, in the strictest and narrowest sense of the term—a reflex inhibition, probably in the majority of cases general, affecting all the functions of the nervous system, and not limited to the heart and vessels only."

Pathology of collapse.—It has been pointed out already that collapse occurs in the course of those diseases in which the body loses a large

quantity of fluid at a time when vomiting is interfering with the intake of fresh supplies.

The appearances presented by the tissues of a person who has died of Asiatic cholera clearly reveal the effects of this loss of fluid. The spleen, lungs, and liver are tough and leathery; and the blood which is found in the heart and vessels is so remarkably viscid that it has been said to resemble tar (6). The same change in the blood has been detected by venesection during life. The state of the organs after death, taken together with the shrunken appearance of the features which is so marked while the patient is still alive, points very strongly to the conclusion that various solid tissues are called upon to supply fluid to take the place of that which has passed from the blood into the alimentary canal. And this opinion is supported by the fact that during the attack of cholera dropsical effusions have been known to disappear in a few hours. On the other hand, the thickening of the blood itself shows that this compensatory flow of fluid from the solid tissues is not sufficient to make up the loss.

The changes in the blood and organs just described are not peculiar to cholera; they were seen by Hilton Fagge in a person who died of acute ulceration of the intestines; and it is reasonable to infer that they are present in all patients who suffer from severe and prolonged diarrhœa and vomiting.

On the other hand, it is not so obvious that the collapse which may follow abdominal operations and which accompanies peritonitis is associated with a similar inspissation of blood and tissues. For although the appearance of the patient suggests such a change, yet there is no evident loss of fluid sufficient to account for it. Experiment, however, has not only made it certain that under these conditions the blood and tissues do lose much of their water, but has also revealed what becomes of the fluid which they lose.

The first observers to throw any light upon this question were Professor Sherrington and Dr. Copeman, who observed that after opening the peritoneal cavity in rabbits the blood rose considerably in specific gravity. This observation was followed by a series of experiments made by Professor Roy and myself. As these experiments have never been fully published, I shall give a short description of them here: this I do with the full consent of Professor Roy. Dogs were placed under the influence of an anæsthetic, and kept so during the whole course of the experiments, which often lasted from twelve to eighteen hours. Collapse was brought about by opening the abdominal cavity widely and disturbing the intestines, which, as a rule, were divided between ligatures. A continuous graphic record of the blood-pressure was obtained by means of a manometer connected with the carotid artery; the specific gravity of the blood was measured at intervals of about one hour. In order to follow the distribution of fluids in the injured and uninjured tissues respectively, the specific gravity of the intestinal wall and of the skeletal muscles also was

observed at the same time. The muscles were selected as a type of the uninjured tissues, because it was easier to observe their specific gravity than that of other tissues; but it is almost certain that alterations similar in character to those observed in the muscles were taking place in other parts, in some possibly even to a greater extent.

The changes observed in the course of these experiments were as follows:—From the time the abdomen was opened the specific gravity of the intestinal wall diminished, and that of the skeletal muscles increased very considerably; but the specific gravity of the blood for an hour or two did not show any marked change. These rapid alterations in the density of solid tissues could only have been produced by the gain or loss of a fluid of a specific gravity different from their own. It is possible that they were caused by alteration in the distribution of the blood in the tissues in question. But the specific gravity of the blood does not differ much from that of these solid tissues, and the changes appeared to us to be too great to be explained in this way: it seemed more probable that they were caused by alterations in the distribution of the lymph. This fluid appeared to pass out of the blood into the injured peritoneum and subperitoneal tissue, while for a time the blood made good its loss by taking up fluid from the muscles and, presumably, from other uninjured tissues also. So long as this compensation was complete the circulation remained undisturbed. But at length a moment arrived when the reserve of fluid in the uninjured tissues was exhausted, and after this moment there was practically no further rise in the density of the muscles; but the specific gravity of the blood began to increase, and thenceforward steadily continued to do so, until the blood was so much thickened that it flowed with difficulty from any but the largest arteries: in some experiments, indeed, after the lapse of twelve or eighteen hours, it attained a specific gravity higher by as much as 14° than at the beginning of the observations.

For some hours after the specific gravity of the blood had begun to rise the arterial pressure showed little or no sign of failing, but the pulse gradually became more rapid and smaller. When at last the blood-pressure began definitely to fall it continued to do so rather rapidly, and death usually occurred a few hours later. The temperature during the course of the experiments showed a marked tendency to fall; it was, however, maintained at its normal level by suspending the animals over a bath of warm water, maintained at the necessary temperature.

The events which occurred in these experiments as the result of a severe abdominal operation may be arranged in three periods:

(*a*) During the first period fluid was poured out into the injured tissues; but the blood remained unaffected by this loss, because an equal quantity of fluid was passing into it from the uninjured tissues.

(*b*) During the second period this compensatory flow of fluid became insufficient to meet the loss, the density of the blood gradually increased, and signs of failure of the circulation began to appear; nevertheless the arterial pressure remained practically unchanged.

(*c*) The third period was that of the fall of blood-pressure, and it continued until the death of the animal.

The duration of the first period varied in different cases from one to several hours. It probably depended partly upon the severity and extent of the injury, and partly upon the quantity of fluid present in the tissues of the animal at the beginning of the experiment.

As the uninjured tissues were able to supply the blood with fluid to compensate the outflow into the injured tissues, it is very probable that in health the tissues have the function of reservoirs, and give fluid back as may be required to maintain the specific gravity of the blood at a constant level.

While during the course of each of these experiments the specific gravity of the blood increased enormously, that of the plasma underwent even a slight diminution. This observation pointed to one of two conclusions: either that the fluid which left the vessels was of greater specific gravity than that of the plasma within, or that the fluid which poured into the blood from the muscles was of lower specific gravity than the plasma; the latter is by far the more probable conclusion, for we know that the density of lymph is normally less than that of plasma.

The loss of fluid suffered by the blood during one of these experiments may be calculated from the change recorded in the specific gravity of the latter, if we assume the specific gravity of the fluid lost to be the same as that of the plasma; and this could be directly measured. In many of the experiments an additional method of estimating this loss of fluid was employed. Small quantities of blood were taken at intervals of two or four hours from the femoral artery and mixed with some oxalate of potash solution to prevent clotting; at the end of the experiment these specimens were whirled in a centrifugal machine, and after this had been done, the actual volume of corpuscles and plasma could be seen and measured. This method showed a loss of fluid which closely agreed with that estimated from the observed changes in the specific gravity of the blood, and revealed the astonishing fact that sometimes the blood might lose as much as one-third of its original volume, while presumably it still contained the same number of corpuscles. No wonder that thus thickened it passed through the smaller vessels with difficulty.

During the whole course of each of these experiments the dog was kept under the influence of an anæsthetic, and of course received no water. It might be thought that this management alone would account for a rise in the specific gravity of the blood; but control experiments showed that these animals may be kept for many hours under conditions which only differed in that the abdominal cavity was not opened, without any notable change in the specific gravity of the blood.

A well-marked increase in the density of the blood of dogs after opening the abdomen has been observed by Professor Sherrington also (16), who measured the changes which it underwent in the course of some operations which were being performed by Messrs. Ballance and

Edmunds. The principal object of these operations was to ascertain the best method of uniting the divided intestine; antiseptic precautions were therefore used, and, as in the case of like operations on man, every care was taken to do as little damage as possible.

In human beings also, under similar conditions, Dr. Grünbaum has observed a rise in the specific gravity of the blood. He has kindly allowed me to refer to some hitherto unpublished observations. In three instances of laparotomy, in which the operation lasted about an hour, the rise was considerable, and varied from five to seven degrees; while in some others of short duration it did not exceed one or two degrees.

It is important to point out that the changes in the density of the blood observed in these latter instances were not delayed as in our own laboratory experiments, but occurred during the course of the operation. A possible explanation of this difference is afforded by the fact that patients who are prepared for abdominal operation are not allowed to take any liquid for some hours before. The object of this precaution is to diminish the risk of vomiting; but after what has been said, it is probable that, if carried too far, it may be a distinct disadvantage, and cause the reserve of fluid in the tissues to be materially diminished at a time when it is to be called upon for the purpose of maintaining the density of the blood at its normal level.

There can be no doubt, then, that after a severe abdominal operation the blood may lose much of its fluid and become thickened, as it does during an attack of cholera. Is it unreasonable to attribute to this cause the symptoms of collapse which often follow such operations? Long ago it was pointed out by Cohnheim (4) that the thickening of the blood in cholera is sufficient to account for the fall of arterial pressure, the diminished size of the pulse, the retardation of the entire circulation, and the wan appearance of the patient. And what the present experiments and observations have done is to establish the fact that collapse which may follow abdominal operations is associated with changes in the blood similar to those which occur in cholera and other kinds of diarrhœa. And we cannot doubt that the collapse of peritonitis is due to the same cause.

To these lesions we must add one other which also causes the density of the blood to increase. I refer to the inflammatory œdema caused by burns and scalds. Sherrington (16) has recently shown that immersion of the hind limbs of animals in hot water for several minutes is followed by a very marked rise in the specific gravity of the blood, which reaches its maximum in a few hours, and continues at that elevation, with but slight diminution, for two or three days. In these animals full anæsthesia was maintained during the experiment, and the nerves of the limb or limbs in which inflammatory œdema was caused were divided to prevent subsequent pain; consequently afferent impulses from the seat of injury were prevented from reaching the nervous centres and affecting the circulation by reflex action. No doubt

when burns and scalds occur while the nerves are intact such impulses do exert a considerable influence; and we must regard the prostration which results in these cases as partly due to reflex nervous inhibition, partly to inspissation of blood. Indeed I do not intend to deny that in abdominal operations, and perhaps also in diarrhœa, especially if pain be present, such nervous impulses may play a part.

There are two other accidents capable of causing profound prostration which have not yet been mentioned. I refer to the twisting of the pedicle of an ovarian tumour, and the perforation of the stomach or intestine. These accidents would seem to be capable of causing prostration, partly through the agency of the nervous system, and partly also by causing œdema of the organ affected by the torsion in the one case, or of large areas of inflamed peritoneum and subjacent tissues in the other, and so leading to an increase in the density of the blood. Indeed in clinical practice collapse and shock are often associated.

Moreover, the fact already referred to, namely, that certain poisons are capable of causing prostration without having produced effects likely to alter the physical constitution of the blood, forces us to recognise that collapse, even in one of its simplest forms—when, for instance, it occurs in the course of severe diarrhœa—may be due, not entirely to changes in the blood caused by loss of fluid, but also in some part to the constitutional action of poisonous substances produced by micro-organisms in the alimentary canal and absorbed into the blood. But the part played by these poisonous substances in causing collapse is probably of secondary importance. And it cannot be doubted that, of the two main varieties of prostration, one is due to the effect produced upon the nervous system, either directly by emotion, or by means of afferent impulses from an injured part, and the other to alterations in the physical constitution of the blood. The former includes all that which has hitherto been called shock, while the latter includes at least all the more important examples of prostration to which the name collapse has been given.

Prevention of shock.—It is well known that amputations for disease are borne better than those for injury, and that persons in vigorous health are often bad subjects for operation. This may be because those who suffer from disease become, in the course of time, resigned to their misfortunes, and are therefore less affected by the thought of losing a limb than are those who have previously enjoyed good health. But it is probable, also, that confinement to bed and keeping the body in a constrained position is less easily borne by those previously healthy than by others who are more used to the conditions imposed by ill-health (17). It is an excellent practice, therefore, whenever an operation likely to be followed by shock is to be performed, to keep the patient in bed for several days beforehand.

Treatment of shock.—Since shock is partly due to failure of the circulation brought about by loss of tone of the abdominal blood-vessels, the position of the patient is of great importance. This was clearly

shown by Goltz's experiment on the frog, in which, after palsy of the abdominal vessels by striking the surface of the abdomen, the body of the animal was opened. When the heart, which has ceased for a few seconds, begins to beat again, it is observed that as long as the animal is held with its feet down no blood enters the heart, but does so as soon as its position is reversed.

More recently Mr. Leonard Hill (9), from numerous experiments on larger animals, has drawn the conclusion that "the important duty of compensating for the simple hydrostatic effects of gravity in changes of position must be ascribed to the splanchnic vaso-motor mechanism, that when the power of compensation is damaged by paralysis of the splanchnic vaso-constrictors induced by severe operative procedures, by injuries to the spinal cord, or by such poisons as chloroform and curare, then the influence of gravity becomes of vital importance." Patients suffering from shock should therefore be kept in the horizontal position, with the head low and the limbs raised. In this way the return of blood to the heart is facilitated and its intake increased. This result may be largely augmented by tightly bandaging the abdomen. If the heart is working normally, these procedures will raise the blood-pressure to the level of health; but when its activity is reduced, as it is in shock, there is some danger that they may embarrass its action by causing engorgement of its right side. Bandaging of the abdomen should be therefore resorted to in a tentative manner, the effect upon the pulse being watched meanwhile.

It is important that the patient be kept warm, and with this aim he should be wrapped in hot blankets and a hot-water bottle be placed at his feet. Hot baths have been given for the same purpose with good effect, especially to children suffering from burns.

Alcohol, ammonia, and other stimulants are of great service in milder cases, and opium is useful to relieve pain and excitement. Nitrate of amyl, atropine, strychnine, and inhalations of oxygen have been used without much effect. Digitalis has been given by Dr. Wilks (19) with benefit to the patient, and is considered by Dr. Lauder Brunton to be worthy of a much more extensive application in cases of shock than it has hitherto received. In no case must drugs be pushed, for the arrest of absorption and possibly also of the vascular exchanges may delay their action; and when reaction sets in, if previously given in large quantities, they may produce serious symptoms of poisoning.

Treatment of collapse.—In those cases of prostration in which the symptoms may reasonably be attributed to inspissation of the blood the intravenous injection of normal salt solution would seem to be indicated. Nevertheless, in spite of the wonderful immediate effect of this procedure upon patients suffering from the collapse of cholera, the benefit is but transitory, and it is doubted by good authorities whether the mortality of this disease has been reduced by it.

In the experiments made by Professor Roy and myself, the intra-

venous injection of salt solution into dogs suffering from collapse served only to reduce the specific gravity of the blood for a short time; the specific gravity rapidly increased after the injection, and in an hour or more was as high as before. A solution capable of producing a more lasting effect is greatly to be desired. Sherrington and Copeman have shown that injections of strong sugar solutions answer this requirement better than injections of weak salt solution. This plan of treatment, therefore, offers some prospect of success, but further experimental evidence of its value is required before it can be recommended as a method.

In cases of collapse from diarrhœa water should be freely given by the mouth. After abdominal operations and in peritonitis, it is often necessary to limit the amount of fluid given in this way on account of the risk of encouraging vomiting; in these cases rectal injections may often be given with advantage. In preparing patients for abdominal operations it is the custom to keep them without water for some hours beforehand. It may reasonably be doubted whether this plan is altogether beneficial, for it seems calculated to increase the risk of subsequent collapse. On the other hand, it must be remembered that under these circumstances fluids swallowed increase the exudation into the peritoneal cavity, and consequently the risk of peritonitis. It is possible that, after abdominal operations, escape of fluid into the peritoneal cavity and tissues may be lessened by means of a carefully applied abdominal bandage.

In other respects collapse should be treated on the same general principles as shock. Diffusible stimulants should be given, and the bodily temperature kept up by hot applications.

LOUIS COBBETT.

REFERENCES

1. BARLOW, LAZARUS. *Proc. Physiol. Soc.* May 12, 1894; *Jour. of Physiol.* vol. xvi.—2. BRUNTON, LAUDER. "Pathology and Physiology of Shock and Syncope." Read before the Abernethian Soc., St. Bart.'s Hosp.—3. *Idem.* "On the Employment of Nitrate of Amyl in the Collapse of Cholera," *Brit. Med. Journ.* June 13, 1872.—4. COHNHEIM. *Lectures on Gen. Pathology.* New Syd. Soc. vol. cxxvi.—5. CONTEJEAN. *Archives de Physiol.* 5me série, tom. vi. 1894.—6. FAGGE. *Principles and Practice of Medicine*, 1st edition, vol. i. pp. 15, 297, 298.—7. FOSTER. *Text-Book of Physiology*, 5th edition, p. 325.—8. GOLTZ. *Virchow's Archiv*, vols. xxvi. and xxix.—9. HILL, L. *Jour. of Physiol.* 1895.—10. HORSLEY, VICTOR. Heath's *Dictionary of Surgery.*—11. HUNTER, JOHN. Works edited by Palmer, vol. iii. pp. 91 and 114.—12. MACCORMAC. "Shock and Collapse," Quain's *Dict. of Med.*—13. MOULLIN, MANSELL. "Shock," Ashurst's *Encyclopædia of Surgery.*—14. ROGER. *Archives de Physiol.* 5me série, tom. v. 1893; and tom. vi. 1894.—15. SHERRINGTON and COPEMAN. *Jour. of Physiol.* vol. xiv. 1893.—16. SHERRINGTON. *Proc. Roy. Soc.* April 1894.—17. TRAVERS. *Constitutional Irritation.*—18. WATSON, CHEYNE, and TREVES, C. *System of Surgery.*—19. WILKS. *Med. Times and Gazette*, January 1864.

L. C.

DISEASES OF THE MOUTH

Catarrhal Stomatitis (Acute stomatitis, Simple stomatitis, Erythematous stomatitis, Pultaceous stomatitis) is the mildest form of inflammation affecting the mucous membrane of the mouth, but it is one which often forms the starting-point for some of the severer kinds of stomatitis.

It occurs most frequently in children, but adults are also liable to it. The appearance of the mouth depends on the extent of the mucous membrane involved. The inflammation may occur in patches which may present an erythematous appearance; in this case the surface will be drier than normal: or the patches may reach the catarrhal stage, with some thickening, and marked increase in the secretion of the mucous membrane. If this latter stage persist, even for a few hours, the mucous membrane will probably appear whitish from the presence of partially shed epithelium.

If the inflammation be more severe it may extend to the underlying tissues, producing thickening and stiffening of the cheeks, lips and tongue, with some consequent enlargement of the neighbouring lymphatic glands.

Symptoms.—These are such as might be expected: pain, heat, stiffness, discomfort and dryness of the mouth, giving rise to some difficulty in swallowing. The infant is fretful, dribbles at the mouth, and is unable to suck; so that it soon becomes wasted and feeble. In the adult, in the erythematous stage, the dryness of the mouth gives rise to difficulty in mastication; in the catarrhal stage there is viscid and ropy mucus secreted, with some fœtor of the breath. There is frequently slight fever. Impetigo of the lips, vomiting, diarrhœa, and perhaps convulsions may occur secondarily in children.

Etiology.—The local causes vary with the age of the patient; in the infant, food too hot or too pungent, prolonged sucking at a coral, imperforate teat or ill-developed nipple, uncleanliness of feeding-vessels, hare-lip or cleft palate rendering the act of sucking ineffectual, are common sources of this affection. Later the various disorders of dentition come into play; and, in adults more especially, the excessive use of tobacco, stimulant foods and liquors, and irritation from carious teeth. Applying equally to all ages are the local effects produced by drugs; such as mercury, arsenic, lead, iodine, and bromine. Catarrhal stomatitis also occurs as a result of gastric and intestinal disturbances, and in the course of certain acute specific diseases, chiefly scarlet fever and measles; and from oral respiration enforced by nasal and post-nasal obstruction.

Diagnosis is easy, and, if reasonable care be taken, the course is almost invariably towards recovery, in about a week. Infants, however, if they are unwilling or unable to swallow their food, may be in danger

of starvation. If the cause—such as dirty bottles and teats—be not removed, the disorder is likely to recur.

Treatment consists in strict cleanliness, careful feeding, and the use of such antiseptic mouth-washes as solutions of boric acid (2 to 5 per cent), sodii bicarb. (5 per cent), or chlorate of potash (3 per cent), preceded by washing out the mouth with warm water. If there be much pain, ice may be sucked, or a very dilute solution ($\frac{1}{2}$ per cent) of cocaine may be brushed on. Saline aperients and the alkaline gentian mixture are often useful.

Under this heading may be mentioned foot-and-mouth disease, which occasionally affects human beings (31). It occurs, in both adults and children, as a catarrhal inflammation of the mouth, strictly limited to the gums, lips, soft palate, and edges of tongue. For a full account of this malady the reader is referred to the article devoted to it in the second volume of this *System*.

Ulcerative Stomatitis (Phlegmonous stomatitis, Phagædenic gingivitis, Stomacace, Putrid sore mouth) is an affection of very varying severity; slight cases hardly need any treatment, whereas grave attacks entail very serious risk to life.

Symptoms.—The ulceration usually appears first on the margin of the gums in the region of the molar teeth; more often, it is said, on the left side than on the right. The gums at first are red and swollen, bleed easily, and stand off from the teeth, leaving them bare to the socket. This swollen mucous membrane soon begins to break down, forming a dirty yellow or gray ulcer. By this time the neighbouring surface of the cheek and tongue will probably be affected also, and the ulceration may spread round to the inner surface of the lips. In severe cases the cheeks and tongue are swollen and stiff, and move with pain and difficulty. The tongue becomes marked on the edge by the teeth, and its surface is thickly furred. The teeth left bare by the gums are apt to be loosened, and even to drop out. There is increased secretion of saliva, which dribbles from the mouth during sleep. The breath is very offensive. The submaxillary glands are almost always enlarged.

Pathology.—This affection is histologically one of molecular necrosis, with surrounding small-celled infiltration. The ulceration differs from that of gangrenous stomatitis in that it tends to spread superficially, not deeply. The question of the existence of a definite micro-organism is still unsettled, but the character of the disease certainly suggests such a cause, and the fact that Bergeron inoculated himself successfully (19) with this affection further bears out the idea.

Ætiology.—This disease occurs most frequently in children between the age of four years and puberty; and is usually due to one or both of two causes—insanitary surroundings and some local irritation, such as a decayed and sharp tooth. Mercurial poisoning is another cause. Overcrowding appears to favour its spread. There is no doubt that it is largely due to improper feeding, but in older children this is more difficult

to ascertain. The condition of the teeth is certainly a very important element in its production, as it never appears before the teeth.

The *duration* of this affection varies with its severity; if properly attended to, it usually subsides in the course of ten days, even in bad cases: the fœtor of the breath, indeed, is so great that such cases usually receive early treatment.

Treatment. — Fortunately in chlorate of potash we have an efficient remedy which rarely fails. Its action is both local and constitutional, for the salt when swallowed is excreted unchanged by the saliva. Perhaps the best form of administration, in order to secure both these actions, is in compressed tablets which may be sucked and the saliva swallowed.

If the treatment can be safely carried out, it is well also to apply to the affected parts on a cotton wool pledget a strong solution of the perchloride of mercury, made up as follows: Liq. hydrarg. perchlor., Glycerin., Aquæ rosæ āā ʒij., care being taken, of course, that the solution does not run down the throat, as salivation might easily be thus caused. Various other antiseptic applications (such as those suggested in the preceding section) are also useful. To avoid the mechanical irritation of solid food the child must be fed on slop diet.

In three or four days the ulcerated surface thus treated should be clean and healing, and in a few days more recovery should be complete.

Bednar's aphthæ (*aphthæ of the palate—plaques pterygoidiennes*).—This form of ulcerative stomatitis must be separately described. This affection occurs in weakly children who are bottle-fed, and appears to be due to the pressure of the teat or the nurse's fingers against the palate: this causes a local mycotic necrosis of the mucous membrane, originating in the invasion of bacteria into the superficial epithelial layer of the palate. The gradual detachment of the necrotic epithelium gives rise to ulceration and permits the entrance of more micro-organisms. The infection may become general through the medium of the lymphatics (21, 34). The prognosis is generally favourable, and if the cause be removed no other treatment is necessary.

The so-called *simple ulcers* of the mouth are patches of superficial ulceration occurring usually on the inner surface of the lips. Payne and Sevestre have drawn attention to the association between this condition and a pustular eruption on the hands and face (impetigo), and think that both affections are probably due to the same infective process; in further proof of this Gaston and Solis Cohen (37) state that in these ulcers they have found staphylococcus aureus always and almost exclusively. The disorder is almost confined to badly-nourished and feeble infants.

In children an ulcer often forms on the frænum linguæ as the result of the contact between the frænum and the lower incisors in the violent paroxysms of whooping-cough.

The mucous membrane of the mouth is sometimes extensively affected by the contact of hot or caustic substances, such as boiling water or a mineral acid. These produce a sudden superficial necrosis of the mucous

membrane, rendering it white and sodden, and giving rise to the appearance of a "membranous" covering. When this falls off the deeper epithelial layers are denuded of their covering, and a surface is left which readily bleeds. The same appearance may occur in diphtheria; the membrane —having the usual appearance of diphtheritic exudation—may either affect the lips primarily, or secondarily the tongue, cheeks, lips, or gums. It is comparatively rare, however, for the mouth to be attacked by diphtheria, especially as the primary seat. Where the diagnosis is doubtful cultivation tests must be made.

Pemphigus vegetans (11).—This rare affection is a very serious and fatal form of pemphigus, beginning in the mouth, palate, and pharynx. Pain on eating and swallowing is the first symptom, and patches of the mucous membrane of the tongue and cheek are found to be white and more or less detached. After some days or weeks the bullæ of pemphigus appear on the hands and body, run a chronic course, and ulcerate deeply. The pain of eating seriously interferes with nutrition. If the skin be extensively involved the prognosis is invariably fatal. Cases have been reported from all parts of the world. Hutchinson states that some cases recover under opium. Jacobi reports several such cases under the name of Stomatitis neurotica chronica; from the small number of recorded cases he thinks the condition must be very rare: he points out that it seems to occur in neurotic subjects, and that there is usually an hereditary predisposition.

Aphthous Stomatitis. — (Aphthæ, Canker, Vesicular stomatitis, Herpetic stomatitis).

This form of stomatitis is characterised by the presence of numerous small yellowish or grayish patches or superficial erosions surrounded by a slightly reddened base, producing no symptoms beyond slight pain on contact with any sharp or irritating particles of food.

Pathology.—The essential lesion consists in little papules, which may be single or multiple, and may occur in any part of the mouth, but most commonly on the inner surface of the lower lip near its junction with the gum. The papules are due to local hyperæmia and cellular proliferation. At the end of twenty-four hours they lose their epithelial covering, and there is seen a little oval, creamy or whitish patch level with the surface of the mucous membrane, although the edges may be somewhat raised from infiltration. This patch disappears in a few days and leaves a sharply-defined erosion somewhat conical in shape. Although it is often called an ulcer, Bohn points out that, as there is no pus formation, this name is not accurately applied to it. The phenomena suggest a parasitic cause, but no organism has yet been detected.

Under ordinary circumstances the sore heals in a few days by the spreading of a new epithelial covering from the margin.

Some catarrhal stomatitis in other parts of the mouth is often associated with it.

Ætiology.—This affection is more common in children and is dependent on the usual causes of stomatitis, imperfect hygiene and careless feeding. In adults a single aphthous spot sometimes appears in persons apparently quite healthy; possibly in such cases it is due to some local irritation.

There can be no difficulty in the *diagnosis*, and the *prognosis* is quite favourable, though in debilitated persons the disorder may last some time. The usual duration is about seven days.

Treatment.—The constitutional state must be carefully attended to, and with children care must be taken also that they do not refuse their food, on account of the pain caused by eating. Strict cleanliness must be observed, and the mouth washed out after taking food, preferably with a weak solution of boracic acid (gr. v. ad ℥j.) or chlorate of potash (gr. v. ad ℥j.) The sores may be touched with a fine point of sulphate of copper or nitrate of silver (care being taken to avoid injuring the surrounding mucous membrane). Permanganate of potash (1 per cent solution) or Liq. hyd. perch. may be painted on the spots. If there be much local pain a 3 per cent solution of cocaine may be applied before the meals.

It is well to begin treatment with a saline aperient; and chlorate of potash may be given internally, as in ulcerative stomatitis.

Parasitic Stomatitis.—(Mycotic stomatitis; Thrush, Soor, Muguet.)

Possibly the restriction of the name "parasitic stomatitis" to cases of thrush is hardly accurate, as undoubtedly some of the other forms of stomatitis are due likewise to the existence of micro-organisms; but thrush is such a well-recognised condition that it must be discussed separately.

Bacteriology.—Shortly, the disease is catarrhal stomatitis having associated with it a fungous growth which gives rise to the characteristic white patches of varying size on the dorsum of the tongue and inner surface of the cheeks and palate.

The fungus was described as long ago as 1843 by Berg, and shortly after by Robin, as oidium albicans. Since then it has been stated by Hallier to be oidium lactis; by Grawitz to be mycoderma vini; by Rees to be saccharomyces albicans; and by Plaut to be monilia candida: on the whole, however, opinions lean to the oidium albicans. Probably the explanation of the discrepancy lies in the remarkable polymorphism of this fungus under varying conditions of cultivation, variations which have been studied by Audry. Briefly, it may be stated that the more solid the media of cultivation, the greater the tendency to spore-formation; the more liquid the media, the greater the tendency to filament-formation. It may well be that the varying condition of the mucous membrane of the mouth may result in the predominance of one or other of the varieties.

Pathology.—The changes apparent in the mucous membrane are those of catarrhal stomatitis; namely, redness, and diminution in the buccal secretion: soon one or more small white spots the size of a pin's head appear, which very much resemble flakes of curdled milk, but require

considerable force to detach them owing to their being covered by a layer of epithelium; when detached a raw surface appears, which bleeds readily. The spots are due to the spores which develop between the epithelial cells, and then spread upwards to the surface and downwards to the connective tissue. The superficial filaments then tend to spread over the surface of the mucous membrane, and thus larger discrete patches are formed which may finally coalesce into a layer almost uniform all over the tongue. In this later stage the deposit, consisting partly of fungus and partly of cell débris, can be more easily detached. There is no change in the mucous membrane itself beyond what was described above.

The *symptoms* are often so slight that the existence of the affection is only found out by chance. They are the same as those produced by catarrhal stomatitis, but in this case some diarrhœa or vomiting is more frequently associated with the mouth affection.

Ætiology.—The causes of this affection are uncleanliness, debility, and the existence of catarrhal stomatitis. It is met with most commonly in infants, next in extreme old age at the approach of death; but it may occur at any age in states of great debility; such, for instance, as are produced by cancer, tuberculosis, or enteric fever. One condition is present in all these cases, namely, restricted movement of the tongue and mouth, which no doubt permits the lodgment and growth of the fungus; especially as this restriction is often associated with oral respiration, which must facilitate the entry of the spores which are always present in rooms or wards where other patients are or have been suffering in the same way. Catarrhal stomatitis, by causing a loss of vitality in the superficial layers of the epithelium, also favours the microbic growth. The acid condition of the buccal secretions, so often regarded as favouring the growth of the fungus, seems to be rather a result than a cause.

Lastly, the most common sources of infection are dirty feeding-bottles, spoons, feeders, and other articles; particularly when milk is the main article of diet. The infection may even be transmitted from one infant to another by the mother's nipple.

The situations, where the growth most commonly occurs, are the dorsum of the tongue, the inner surface of the cheeks, and the soft palate; but it may spread to the pharynx, and even down the œsophagus to the stomach: the ciliated mucous membrane of the nose and larynx are never affected.

There is a popular and strongly-rooted notion that "the thrush passes through children" and emerges at the anus. The evidence of this extension is not satisfactory, and the probable explanation is that the general digestive disturbance associated with the existence of thrush results in an attack of diarrhœa which produces some excoriation of the anus and nates. It is remarkable how easily an account of buttock excoriation may be obtained in support of a doubtful history of congenital syphilis by asking "whether the thrush has passed through the child," but its reliability as evidence of syphilis appears to me very doubtful. The extremely frequent occurrence

of thrush among the children of the poor, as a result of ignorance and dirt, is more clearly brought out by such questioning.

There can be very little difficulty about the *diagnosis* of this condition; small patches of adherent milk-curd are the only things likely to be mistaken for it. The microscope affords a certain means of settling the question.

The *prognosis* of the disease itself is quite favourable. Healthy children will soon recover from it, perhaps even without treatment. In feeble children and adults it may last much longer, and its not infrequent occurrence in moribund persons has led to its being regarded as a more serious affection than it really is. Its severity is generally commensurate with the extent of the deposit.

Treatment is simple enough—it is practically summed up in the word "cleanliness"; still many people appear to find this a difficult condition. As a preventive, in artificial feeding, all milk should be sterilised or boiled, and the bottle always scalded before use. If the child be suckled the mother must attend to the cleanliness of her nipples.

If the disease already exist, the fungus should be wiped off every two or three hours with a piece of soft linen either dry or soaked in boric acid solution, and the mucous membrane then brushed over with a solution of boric acid, salol, perchloride of mercury (1 to 4000), hyposulphite of sodium (gr. xx. ad ℥j.), borax or permanganate of potash; or a weak alkaline solution may be employed.

The necessary treatment must at the same time be directed to the constitutional condition of the patient. Alcoholic stimulation may be necessary.

Gangrenous Stomatitis (noma, cancrum oris). — This variety of ulcerative stomatitis, which is usually described as a separate disease, is fortunately rare; only one instance has occurred during the last ten years in the records of Westminster Hospital (47). The prognosis is a graver matter by far than in the other forms of stomatitis.

Ætiology.—The causes of noma are various, but some previous deterioration of the health, whether due to imperfect hygienic surroundings, starvation, malaria (a common cause in India), or some chronic or acute disease, is almost invariably present. Of the latter, measles in the first place, and enteric fever in the second, are much the most frequent precursors. It occasionally follows ordinary ulcerative stomatitis.

It is more common in damp countries, such as Holland, and in the spring and autumn. It almost always occurs in children between the ages of two and five.

No organism has yet been found whose connection with the malady can be looked upon as invariable; though, as several members of a family have sometimes been attacked together, there is some evidence of infection. Rauki and Lingard have both described associated organisms, but that they were causative agents is not clear. Dr. Sansom described certain highly refracting bodies which he found in the blood, urine, fæces,

and discharge from a case on the third day of the disease. They were like colourless crystals, $\frac{1}{20}$th the size of red corpuscles, with spontaneous movement which was influenced by chemical agents. The blood injected into mice produced septicæmia and death, and there was a multiplication of these bodies in the blood of the mice; but in the case of a cat, although fatal peritonitis was induced by injection of the discharge into the abdominal cavity, no such bodies could be found; nor could they be found in a second case of human cancrum oris investigated by Sansom.

The objection to the view that noma is a local gangrene, due merely to the low vitality of the child, is that the cheek is from anatomical considerations the last place likely to be attacked, seeing that its blood-supply is very free; in this respect it contrasts markedly with the situation of the other local gangrenes occurring in diabetes, typhus, and other exhausting diseases.

Pathology.—There is some doubt about the earlier changes, owing to the rapidity of the onset and progress of the disease; but probably the first sign is a bleb on the mucous membrane of one cheek, or merely induration of the cheek itself: ulceration rapidly sets in, and in a few hours the whole thickness of the cheek becomes gangrenous, the necrotic process extending from within outwards. Owing to early plugging of the vessels hæmorrhage does not occur. The skin over the internal slough becomes tense and red, then livid and black; an external bleb often forms, and perforation occurs. The necrosis by this time has usually spread to the jaw, the teeth become loose and drop out, and, unless death intervene, the process extends deeply into the lower jaw, upper jaw, and even into the orbit and malar bones. This destruction may occur with great rapidity, even in as short a time as four or five days, or it may be prolonged to a fortnight; but as a rule it is cut short by the death of the patient, either from direct poisoning or from exhaustion caused by diarrhœa probably due to swallowing the fœtid secretion of the ulcer, or from an infective broncho-pneumonia set up by inhalation of contaminated air.

Symptoms.—These are such as naturally follow on the condition described above. But there are several points which need emphasis.

The disease on the whole is remarkably painless. Often the first thing noticed is fœtor of the breath with redness and swelling of the cheek, and examination at once reveals a large sloughing ulcer inside the mouth; but even while the cheek is being perforated and the jaw-bones attacked, the child does not seem to suffer any pain. As the disease progresses, however, the child sinks gradually into an apathetic and delirious state, which passes into one of extreme prostration, ending either suddenly or gradually in death. Secondary broncho-pneumonia often comes on, but, as is usual in this condition, with very little symptomatic indication beyond a little quickening of the respiration.

The fever is moderate throughout, and, curiously enough, there is comparatively little swelling of the submaxillary glands.

It is rare for both cheeks to be attacked, but such cases are on record.

Diarrhœa is almost always present, due probably, as already stated, to swallowing the discharge from the ulcer; sometimes, however, actual ulcerative colitis is present.

Gangrene may also affect other parts of the body, as the lungs, genitals, or limbs; in the latter instances it most likely results from embolic infection.

Prognosis.—The mortality of this disease is very high—about 80 per cent. The prognosis depends on the extent of the disease and the rapidity of its progress. If the patient recover there is almost certain to be considerable deformity of the cheek, and eversion of the lower eyelid.

Treatment must be prompt and decisive, otherwise the rapid progress of the disease and the occurrence of pneumonia will render all assistance useless. The removal of the whole of the gangrenous material either by the cautery or the knife is necessary. The child must be anæsthetised, and the destruction or removal of the diseased tissues be carried well beyond the zone of apparent infection, the utmost care being taken to prevent the inhalation or swallowing of any infected material. So important is this latter point, that it is sometimes thought wise to perform a preliminary tracheotomy (17), and then to plug the posterior part of the mouth with absorbent gauze. The wound and mouth may then be treated in a more thoroughly antiseptic manner. In cutting round the disease it is important, if possible, to save the angle of the mouth, as the resulting deformity will then be less; of course any necrosed bone will be removed at the same time.

If the case be seen early and the disease have not spread to the outer surface of the cheek, it is a common practice to apply nitric acid, or some other equally powerful caustic, freely to the ulcer on the inner side of the cheek, the sloughs having first been scraped and rubbed away. The neighbouring mucous membrane must be protected by the application of oil, and a saturated solution of potassium carbonate must be at hand to control the extent of corrosion. The objection to this form of treatment —which is often carried out as being easier and not so severe—is that the action of the caustic is very apt to be limited through its absorption by the already dead tissue, so that the margin of the disease, which is the important part to attack, is never influenced at all. After any of these operations the surface must be kept as aseptic as possible by frequent applications of a solution of perchloride of mercury or boric acid.

It is most important from the first to maintain the strength of the patient in every way. Plenty of liquid nourishment in a concentrated form must be given at frequent intervals, and alcoholic stimulants are early indicated. In some cases, after surgical measures have been taken, feeding with a nose-tube or by the rectum may be necessary. The child should be kept in the prone position as far as possible, so that the fœtid material may readily escape from the mouth. A steam spray medicated with carbolic acid or sanitas should be kept constantly playing over the bed—for the sake of the nurse, even if useless for the patient; and isolation is, of course, imperative.

Complications such as diarrhœa must be treated in the usual manner.

Various symptomatic appearances of the oral mucous membrane.—Several acute specific fevers produce changes in the mucous membrane of the mouth; these are measles, rötheln, small-pox, chicken-pox, diphtheria, and erysipelas. Some other maladies, such as purpura, scurvy, hæmophilia, Addison's disease, syphilis, or lead poisoning, are or may be accompanied by changes in the mouth. The reader is referred to the several chapters on these diseases in this *System* for the description of these phenomena.

In some cases of congenital heart disease the mucous membrane of the mouth is markedly cyanosed. In anæmia it is one of the places where the general pallor is most apparent; in jaundice the yellow staining soon becomes visible in the mouth.

Tuberculosis and leprosy in rare instances may occur in this situation, but never perhaps primarily.

Too much stress cannot be laid on the importance of observing the condition of the teeth from a medical standpoint, whether it be with regard to caries in adults as the probable cause of dyspepsia, or delayed dentition in children as an indication of improper feeding and rickets. The notching of the upper central incisors in children who have suffered from congenital syphilis is often of great diagnostic use, but this must not be confused with the transverse furrowing and pitting resulting from antecedent simple stomatitis.

I will only enumerate the affections of the mouth which are more distinctly surgical, such as gumboils—an inflammatory condition about the root of a tooth resulting in suppuration; a nævoid condition of a portion of the gum; and a general or local hypertrophy, which may give rise to an actual polypus. This latter must be distinguished from the more serious condition of epulis, which is a growth of similar appearance, but one originating in the periosteum. Lastly, papilloma and epithelioma may occur in this situation.

The lips are occasionally the seat of cracks, or fissures, which are chiefly noticeable from their resistance to treatment. These cracks usually occur in the centre of the lips or at the angles of the mouth. The two main causes of this condition are syphilis and eczema, the latter resulting from seborrhœa, from cold ("chapping"), or from constant wetting and sucking, as occurs in some severe cases of chorea. Herpes labialis, especially when contaminated with pus organisms, may occasionally leave troublesome cracks behind.

The syphilitic form occurs both in infants and adults, and is usually associated with mucous tubercles of the oral mucous membrane. In babies it is one of the commonest and most characteristic manifestations of inherited syphilis, and in adults it frequently appears in the later stages of the acquired disease. In infants the curiously "puckered" appearance of the mouth, resulting from the thickening and stiffness accompanying the cracks in the lips, is one of the earliest signs of the

disease, and helps largely to give the characteristic expression to the child's face.

Owing to the infiltration, the edges of the cracks are often everted, and on this, and the association with induration and probably mucous tubercles inside the mouth, the diagnosis must be based; even in the absence of signs of syphilis elsewhere.

The appearance of these cracks in adults is very similar to that described above, and associated mucous tubercles are generally present. It must not be forgotten that in both cases the discharge from these surfaces is highly contagious.

In persons suffering from eczematous cracks, it is usual to find evidence of eczema or seborrhœa elsewhere on the face or scalp, from which point the affection has spread to the angle of the mouth. When it has reached this situation and fissures have resulted, they may remain open for years, and, when so chronic, may take months to cure; the constant movement and moisture of the lips interfere greatly with the healing process. The cracks often itch in a most troublesome way, and cause much pain on smiling, eating, yawning, and talking; thus the comfort and even the nutrition and health of the patient may suffer to an unexpected extent.

The treatment of the syphilitic form is that of syphilis generally, coupled with the local use at night of some dilute ammoniated mercury ointment (care being taken that the patient do not suck his lips too freely): during the day the skin must be kept supple with simple boric acid or zinc ointment. Every few days the cracks may be painted with a weak caustic, such as chromic acid solution (1 to 100).

The local treatment of the eczematous form must be carried out on similar lines; but it is much more difficult, and hence many changes of remedies may have to be tried before success is attained. It is imperative first to attend to any eczema or seborrhœa of the scalp; neglect of this precaution is almost certain to produce failure in the lip-treatment. In the more chronic and therefore difficult cases it is especially necessary to obtain as much quiet for the angle of the mouth as possible by prohibiting speaking, smiling, yawning, and so forth; and even by making the patient suck his food through a glass tube for a few days. These precautions will also save the lips from incessant wetting, which is almost equally important. Those who have suffered long from this affection are so anxious to recover, that they will usually submit even to restrictions as severe as these.

The lips must be constantly lubricated by day with "cold cream" or other emollient; this is often advantageously combined with sulphur (gr. x. ad ℥j.) or resorcin (gr. v. ad ℥j.), or with lead ointment (liq. plumbi subacet. ♏ xv. ad ℥j. of either white vaseline, lard, or adeps lanæ hydrosus). At night dilute ammoniated mercury ointment may be applied, or the lips may be brushed with a solution of argent. nitrat. gr. x.-xv. in spir. æth. nitros. ℥j. In obstinate cases, strips of Unna's zinc-ichthyol-salve muslin (plaster mull) should be kept on all night, a weak ointment of salicylic

acid in ung. zinci being applied during the day. The usual internal treatment for eczema will be carried out at the same time. It is well to warn such patients to be careful in future never to let any patch of eczema or seborrhœa on the face reach the mouth, but to take active precautions to cure it at once.

Diseases of the Tongue

The diseases of the tongue generally come under the notice of the surgeon rather than of the physician, but the early stages of all the affections require some notice here.

Glossitis (Inflammation of the tongue).—Acute glossitis is the term applied to acute inflammation of the tongue, whether the mucous membrane only be affected, or the parenchyma of the organ also.

Strictly speaking, every case of stomatitis, where the mucous membrane of the tongue is affected, is a case of superficial glossitis; but the term acute glossitis is commonly applied to the inflammation of the body of the organ. This is a rare affection.

Ætiology.—Exposure to cold is often the immediate cause, especially in alcoholic people; or some injury, such as the biting of the tongue in an epileptic fit, or a septic wound from a wasp-sting or a dirty fork; or it may occur as a result of mercurial poisoning or of acute specific illnesses. Not unusually one side only of the tongue is affected (hemiglossitis) (5), and it is possible that all cases of acute glossitis, if seen sufficiently early, would be found to begin in this way. The affection is more common in men than in women.

Pathology.—The inflammatory changes need no special description and usually undergo speedy resolution; occasionally, however, they result in the formation of an abscess.

Symptoms.—These are such as might be expected. Pain is a prominent symptom, and in severe cases there is considerable difficulty in speaking, swallowing, and even in breathing. The swelling sometimes begins near the root, but more often in front at the point of injury—if the cause be traumatic. The swollen tongue may be so much increased in size as to project beyond the teeth and depress the jaw. The surface becomes dry, brown, and even black. The breath is offensive, and the patient is restless and feverish. There is considerable enlargement of the sublingual glands. When the affection begins as hemiglossitis the left side is usually affected, and an elastic lump may often be felt in the substance of the tongue towards the base. Hemiglossitis is usually a milder affection and rarely needs active treatment. This condition lasts for three or four days, and ends either in resolution or in the formation of an abscess requiring early surgical interference. Sometimes local sloughing takes place. In persons suffering from chronic alcoholism hæmorrhage sometimes takes place into the substance of the tongue, to which the term hæmorrhagic glossitis (29) has been applied. This produces a swelling of a dark blue colour in one

or both halves of the organ, apparent both on the upper and lower surfaces. There is usually some bleeding into the mouth, and slight constitutional disturbance. It seems to depend on a degenerated condition of the blood-vessels due to habits of intemperance.

The *treatment* of glossitis is so essentially surgical that here little more need be said than that in the severe cases early incision is usually necessary. In the milder cases a saline purge should be given, followed by chlorate or citrate of potash (gr. x.-xx.) every four hours. An antiseptic mouth-wash will be necessary. Painting the surface of the tongue with 10 per cent solution of cocaine will often give considerable relief in the early stages.

Chronic glossitis may also be either superficial or deep. Superficial glossitis, if localised, is usually due to persistent irritation by a broken tooth, the stem of a tobacco pipe, and the like. If general it is often called "glossy tongue," and is usually the result of chronic dyspepsia or alcoholism.

Pathology.—This condition is due to a subacute inflammation of the mucous membrane resulting in a shedding of the superficial epidermic layers. Microscopically, the changes consist in thinning of the epidermis with destruction of the papillæ and other appendages; in thickening and increased vascularity of the corium, and infiltration of its superficial layers with nuclei and cells (12, 13, 24, 30).

Symptoms.—The appearance of the tongue in this condition is most characteristic. The surface appears excessively smooth and glossy. At one part the smooth surface is red and raw-looking, elsewhere it is whitish as if it had been touched with caustic. In some places the surface may be ulcerated and exceedingly tender to touch or other irritation. In severe cases there may be some swelling of the organ, and indentation of the edges by the teeth. These changes produce pain and stiffness in the tongue, aggravated by all movements in speaking and eating.

Diagnosis is usually not difficult, but the suspicion of syphilis is apt to arise, as the appearances resulting from this disease sometimes resemble chronic glossitis rather closely, particularly when the glossitis is in patches. Careful inquiry into this matter and into the habits of the patient are very necessary.

Prognosis.—The liability of this affection to result in epithelioma must not be forgotten, as it emphasises the necessity for vigilance.

Treatment must be directed in the first instance to the regulation of the diet and habits. Food must be plain and wholesome. Alcohol—especially spirits—and smoking should be given up. The teeth must be attended to if in any way rough; if artificial, they may require readjustment. Regular and scrupulous cleaning of the teeth must be insisted upon.

Dyspepsia and especially constipation must be removed. Finally, mouth-washes containing tincture of myrrh, weak carbolic lotion, or chlorate of potash should be used; and ulcers are to be painted with a weak solution of sulphate of copper, nitrate of silver, or chromic acid (gr. x. ad ℥j.)

In cases where syphilis is suspected it may be well to begin with the administration of iodide of potassium or mercury; but if no improvement take place they should not be long continued, as these drugs seem to have an injurious effect in some non-syphilitic cases (2).

Acute œdema (urticaria) of the tongue.—One of the occasional phases of urticaria, resulting from alcohol, shell-fish, and other poisons, is a rapid and extensive swelling of the tongue, not infrequently associated with œdema of the aryteno-epiglottidean folds. This particular symptom usually appears only in those patients who are prone to urticarial attacks. The buccal urticaria may, however, precede skin urticaria, and hence mistakes in diagnosis may easily occur. It often "migrates" from the throat to the nasal mucous membrane and thence to the eyelids, and may extend downwards to the œsophagus, bronchi, and even to the stomach. Fortunately the swelling, though very rapid and considerable, and hence dangerous from its liability to produce suffocation, partakes of the usual transitory nature of urticarial œdema, and lasts from a few hours only to twenty-four at the longest. The swollen tongue blocks the mouth and protrudes from it, and gives rise to the same discomfort, difficulty in speaking, eating, and breathing, as is present in cases of acute glossitis.

Prompt treatment is necessary; a powerful purge, such as a drop of croton oil, should be given at once, and an astringent cocaine paint (5 per cent) should be applied. If this do not suffice, the protruding tongue must be leeched, or it may be scarified; and if there appear to be laryngeal obstruction, the same process may be applied to the false-cords —if they can be reached. But the possible necessity of tracheotomy must never be lost sight of, as the condition is certainly dangerous to life.

Leukoplakia.—(Leukoplasia oris; Leucoma; Ichthyosis of tongue; Psoriasis of tongue; Keratosis; Tylosis; Smoker's patch.)

This chronic affection of the tongue resembles chronic superficial glossitis in many ways, and in microscopic anatomy indeed some of the milder cases are indistinguishable from that affection; but the more marked cases are characterised by an actual thickening instead of a thinning (as in chronic superficial glossitis) of the papillary layer of the epidermis.

Ætiology.—Leukoplakia is usually due to some long-continued local irritation, such as the constant use of a glass-blower's pipe, the stem of a tobacco pipe or the stream of smoke issuing therefrom, a badly fitting artificial teeth-plate, strong spirits, highly seasoned foods, and, lastly, to syphilis. The disease is much more common in men than in women.

Symptoms.—The appearance presented by this affection is somewhat variable. The tongue is the part most usually affected, but the mucous membrane of the cheeks opposite the line of junction of the teeth, or the inside of the lips, may also be attacked. This condition may be general or limited to the papillary area. The tongue may be smoother than

natural, and the mucous membrane of a delicate bluish white appearance, but quite supple and soft; or the affected area may appear as if daubed with a smudge of dirty white paint with sharply-defined edges. This is of appreciable thickness, and projects above the surface of the tongue. In some spots this coating may appear as if rubbed through, the surface then being red, raw, and very tender, as is the surrounding mucous membrane.

On the other hand, this change may be much more restricted and confined to a spot on the front of the dorsum, where the stream of smoke from a cigar or pipe impinges. This local affection has therefore been called "smoker's patch." The patch is $\frac{1}{4}$ to $\frac{1}{2}$ inch long and $\frac{1}{8}$ to $\frac{1}{4}$ inch broad: at first it is smooth and red or livid, or pearly or bluish white in colour; afterwards it becomes covered by a yellowish white, thin crust, which gets gradually thicker and then peels off. This process repeats itself again and again. If the patch spreads it merges into the condition of general leukoplakia.

The course is very chronic in all cases, and if the affection be extensive recovery is very unusual. This is partly due to the fact that the patients are seldom seen early, as they feel so few subjective symptoms. The chief danger is the liability to epithelioma (24), which is certainly considerable.

Pathology.—There is at first a local hyperæmia followed by infiltration of the superficial layers of the corium with small cells. The papillæ are flattened, and the epithelium becomes thickened and opaque. The patches are at first scarcely raised above the surface of the surrounding mucous membrane, but if chronic they may become considerably raised and even fissured and warty. The likeness to chronic superficial glossitis is obvious, and probably leukoplakia should be regarded as a form of that affection.

Diagnosis.—The appearance is sufficiently characteristic, but scars left by chronic syphilitic affections of the mouth are rather difficult to distinguish from true leukoplakia. Scars, however, are more stationary both in site and character, and there will be more evidence of antecedent ulceration. Smoker's patch and mucous tubercle sometimes resemble each other rather closely. The position and the more rapid formation of mucous tubercles distinguish them from leukoplakia.

Treatment consists in lessening smoking, and in bad cases stopping it; and also in avoiding irritating ingesta, such as alcohol and spices. In local "smoker's patch" the pipe should always be held on the opposite side of the mouth, and any source of irritation, such as decayed teeth or ill-fitting plates, must be removed.

The general health must be attended to and the bowels kept regular. The tongue may be painted once to four or five times daily, according to the severity of the case, with chromic acid (gr. v.-x. ad ℥j. of water) or with a solution of alum (gr. ij. ad ℥j.); or an alkaline wash, such as borax or bicarbonate of soda, may be used. Mel boracis is sometimes useful. Of course if there be any evidence of syphilis iodides must be given (cf. also 3, 6, 40).

Ulcerations of the tongue.—Under this heading may be included simple and traumatic ulcers, aphthous ulcers, mercurial ulceration, and the ulcerations of tubercle, lupus, syphilis, and cancer; but it would be beyond the scope of this article to do more than mention the last four varieties, which are dealt with more fully in surgical text-books.

The tongue from its position is obviously very liable to injury and ulceration; and it will be found that the immediate cause of almost every form of ulceration is injury of some kind or another. Among the usual sources of injury may be mentioned carious teeth, ill-fitting artificial teeth-plates, biting the tongue, and hot or corrosive liquids.

Simple ulcers include various superficial ulcers, mostly due to traumatic causes such as those mentioned above; but they are specially prone to occur in persons suffering from chronic superficial glossitis, and it is the occurrence of these ulcers which is the chief annoyance in this affection. They are characterised by their smooth, red, glazed surface and their chronic course. They are irregular in shape and often very painful. Their treatment has been sufficiently described under the heading of Chronic Superficial Glossitis.

Another variety of the simple ulcer is the dyspeptic or *catarrhal ulcer*, which usually affects the tip of the tongue. It has no definite shape, but is always red and irritable, while the rest of the tongue is coated with white fur. Its connection with dyspepsia is by no means invariable.

Small circular ulcers, resulting from the breaking down of vesicles and pustules, are also found at the tip of the tongue; they are characterised by extreme tenderness. In children, usually, but also in alcoholic adults, or those enfeebled by disease, similar ulcers are sometimes found on each side of the frænum linguæ.

Aphthæ, affecting the tongue, have been referred to under the head of Aphthous Stomatitis.

Those *traumatic ulcers* which occur in a healthy tongue from definite injuries usually affect the tip or border, are of varying length up to $\frac{1}{2}$ or $\frac{3}{4}$ inch, and are covered with shreddy slough. The edges are sharply cut, and the surrounding tongue tissue is œdematous, which gives to the ulcer an eroded appearance. The tongue is furred and the breath offensive. The diagnosis from syphilis, tubercle, and carcinoma is often difficult, but it is less so when the affection is part of a general ulcerative stomatitis.

Another form of traumatic ulcer is that occurring on the frænum in cases of whooping-cough. It is purely mechanical in origin, and is produced by the contact of the under surface of the tongue with the lower incisors, when it is protruded during violent cough. These ulcers, therefore, are never found in infants before dentition. They occur in about 30 per cent of the cases.

Ulceration as a result of *mercurial poisoning* has been already referred to under Diseases of the Mouth. It may occur either in patients taking mercury, or in workmen using the metal for their trade. There is sometimes rather extensive superficial ulceration, not only of the tongue, but

of the gums and cheeks as well. The breath is extremely fœtid, and salivation is marked. Cessation of the poisoning is soon followed by recovery.

In sprue or psilosis (27, 44, 45, 48) ulceration of the tongue occurs. The recognition of the specific nature of this ulceration is by no means easy, as the cases are so rarely seen in England, and the ulceration closely resembles other forms of stomatitis. As the affection is dealt with at length in this volume (art. "Sprue") it is only necessary here to describe the appearances which may be met with in a patient who has been subject to previous attacks, and has been invalided to Europe on this account.

The tongue may be furred at first, but this is soon followed by denudation and destruction of the papillæ, and a smooth, glossy red surface, occasionally fissured, is left, which is intensely sensitive to hot and pungent liquids. The lower lip and cheeks near the angle of the mouth are often raw, tender, and covered with abrasions, leaving on recovery opaque thickening of the mucous membrane which is liable to crack. There is considerable tendency to recurrence.

Tuberculous ulceration of the tongue is rare, perhaps not quite so rare as is sometimes stated, owing to the difficulty in distinguishing it from the more common conditions of syphilis and cancer. Tuberculous ulceration here is usually secondary to tubercle elsewhere. The ulcer is granular, pale in colour, and flabby in appearance; it is covered with yellowish gray mucus, and the edges are bevelled or sharp cut: the outline is often oval, and the borders sinuous; the adjacent parts of the tongue are slightly swollen and pale; this pallor is very characteristic. A recent tuberculous ulcer is superficial, but an old one is deep. The tongue affected with this disease is slowly eroded. Men are more often attacked than women.

The tuberculous ulcer must be distinguished from the syphilitic by the shallowness, the smaller degree of new growth, and the greater enlargement of the lymphatic glands in the former; from cancer, by the greater pallor of tuberculosis, the œdematous but not indurated condition of the surrounding tissue, and the presence of tubercles in the margin, forming small yellow spots. Tuberculosis tends also to occur in younger persons.

Lupus of the tongue is very rare, and is usually secondary to lupus of the face. It has the uneven nodular appearance peculiar to the disease, and is often associated with lupus of the pharynx and larynx.

I shall refer very shortly to the manifestations of syphilis and cancer of the tongue, as they concern the physician in considering the diagnosis of ulcers and nodules which may come under his notice.

Primary syphilitic ulcers are very rare, but when they do occur the tip of the tongue or the lips are the usual sites. The ulcer is small, prominent, and hard, and the submaxillary glands are usually enlarged.

Secondary syphilitic ulcers usually result from breaking down of pre-existing mucous patches, situated on the tip or edge of the tongue, and exposed to injury by the teeth. The ulceration takes the form of a

winding crack of some length, the rounded border of which is raised, white and smooth, whilst the surrounding mucous membrane, for some quarter of an inch, is red. There is seldom any active inflammation, even about the deeper ulcers. In secondary syphilis cracks and fissures also occur along the borders of the tongue from rubbing of the teeth; these are often very painful and distressing. The diagnosis largely depends on the absence of inflammation and the evidences of syphilis elsewhere.

Tertiary syphilitic affections are not dealt with in this place. The usual rules for the treatment of constitutional syphilis must be carried out; locally applications of chromic acid solution (gr. x. ad ℥j.) by a camel's hair brush three or four times a day will be found to give great relief. Improvement rapidly takes place.

The earliest stage of *cancer* is very often hidden in previously existing benign growth; but if it begins anew it takes the form of a blister or of a pimple, which breaks and produces an excoriation soon passing into a definite ulcer, which is often slightly raised. This does not heal but slowly enlarges with marked induration of the base and secondary enlargement of the submaxillary glands. Its diagnosis and treatment rather concern the surgeon.

Wandering rash.—(Geographical tongue; Ringworm of the tongue; Lichenoid; Circular exfoliation; Pityriasis of the tongue; Eczema of the tongue; Desquamative syphilide of the tongue; Marginate exfoliative glossitis; Circulus or Annulus migrans.)

The very multiplicity of the names given to this affection show how much difficulty has been felt in determining its nature. It was first described by Bridou and Gubler about 1872, but little attention was paid to it until the last few years, during which time several investigations have been made into its causation and pathology (4, 7, 20, 23). Even now the causation remains unknown.

Symptoms.—The affection is a somewhat rare one, chiefly affecting children; especially those that are weakly and subject to digestive disturbances. The course of the disease is usually as follows:—A small patch about the size of a pea—smooth, red, and on the same level as the tongue surface or slightly raised—appears on the dorsum towards the tip. This quickly spreads peripherally and forms a circle or oval, leaving the centre smooth and slightly red, whereas the border is yellow and of a definite outline. Several patches usually occur at the same time, and often have a somewhat symmetrical arrangement. In the course of four or five days the circle or circles may have reached the edge of the tongue and crept over the edge on the under surface. Then only the segment of a circle is apparent on the dorsum. When there are several circles, as they increase in diameter they intersect, but in this case the segment of the less definitely marked circle disappears as it meets its neighbour. The outline is not always strictly circular, but may be wavy or indented. There may be secondary circles within the primary. The circles contract and disappear even more quickly than they appeared, but they are extremely apt to recur.

Pathology.—This affection, from its superficial likeness to tinea tonsurans, has been supposed to have a parasitic origin, but there is no evidence of the presence of any specific parasite other than those usually found on the surface of the healthy tongue.

Dr. Dickinson (14) describes the anatomical changes as follows:—The outer ring presents long papillæ, which are tipped with horny epithelium, and often surmounted with the vegetation common to the coating of the tongue. The central bare part displays papillæ which have become denuded, generally down to the Malpighian layer, with complete loss of superficial epithelium. The deepest part of the Malpighian layer remains, but its cells are elongated. Below this there is much vascular injection of the corium and abundance of extruded leucocytes. These are most numerous in the central bare part of the patch, but they occur also to a less degree under the surrounding ring. The Malpighian layer shows general overgrowth, especially of the papillæ. Thus the change appears to belong essentially to the corium, not to the exposed surface; and to originate in the blood-vessels rather than in the epithelium, the changes in which are secondary to those beneath. The visible part of the disease appears to be one or more rapidly expanding foci of inflammation, or at least of injection and extrusion, which begin in the subepithelial part of the skin and carry superficial changes in their wake. The appearances when looked at minutely are more suggestive of some passing disturbance belonging to the blood-vessels than to a parasite, of which the nidus would presumably be in the epithelium. The bilateral symmetry, which is occasionally exact, is suggestive of a constitutional origin rather than a local one. Parrot's account of the microscopical anatomy is nearly the same; though he seems inclined to ascribe a syphilitic origin to the disease, apparently on insufficient grounds. The present view is that the disease is either of central nervous origin, or is due to some constitutional cause akin to that producing the dyspepsia and other associated conditions

There may be said to be a complete absence of *symptoms* due to the affection itself, although salivation and itching have been recorded. The course of the disease is chronic, and there is a great tendency to recurrence; but this seems to wear itself out, as the disease is so much more common in children than adults, and yet has no effect in shortening life.

The appearance of the disease is so characteristic, and the restriction to children so usual, that there is rarely any difficulty in *diagnosis.* Mucous tubercles and leucoma are the only two diseases which might be confused with it.

The constitutional *treatment* must be tonic and strengthening, and any dyspepsia or constipation present must be attended to. Locally, all that is necessary is the application of some mild astringent lotion containing alum, or tannic acid; or some emollient application, such as borax and honey, or chlorate of potash, which is best taken in the form of lozenges.

Black tongue (Nigrities; Parasitic glossitis; Lichenoid; Glossophytia) is a rare affection, but perhaps it occurs more often than

is supposed; it produces no discomfort, and may easily pass unnoticed by the patient.

Symptoms.—It consists of a discoloration, black at the centre and shading off to brown at the edges, which is situated in the middle of the dorsum, in front of the circumvallate papillæ. It begins as a small patch and then spreads over the greater part of the dorsum. This process may take some three or four weeks, or it may last much longer. After a time the coloration gradually disappears from the periphery towards the centre, and there is often some subsequent desquamation.

Pathology.—On the nature of the affection there has been a good deal of dispute (22, 36, 39, 42, 43, 46). Mr. Butlin (8) regards its origin as parasitic, whereas Mr. Hutchinson (25) believes the condition to be almost always due to intentional deceptive staining. Very possibly there may be instances of the latter kind among hysterical subjects, but some of the cases reported are unquestionably of a parasitic nature. In these the filiform papillæ are elongated, and there is an accumulation upon them of micrococcus spores of the kind usually present on the tongue. These add to the size and length of the papillæ, whilst the hair-like projections consist of epithelial cells piled upon each other, with colonies of micrococci between them. Mr. Butlin suggests that they may produce a pigment which gives them their black colour, such pigment formation being a well known property of certain micro-organisms.

The patients are usually out of health, often old and feeble; tonic *treatment*, therefore, is necessary. Locally, applications of 10 per cent solution of salicylic acid or perchloride of mercury may be painted on the tongue, or peroxide of hydrogen may be tried. But the affection is apt to prove very obstinate.

The appearance of the tongue in health.—It is necessary to discuss shortly the varying appearances of the tongue as looked at from a physician's standpoint, and as used by him as indications of the constitutional state of the patient. For this purpose a brief account of the appearance of the tongue in health must first be given. This is very variable in different persons, and in the same person at different times—so much so that no definite standard can be established. The tongue should be firm, and neither too broad and flabby, nor too sharp and pointed. The movements should be thoroughly under control, and the tongue should be protruded sharply, without tremor, and in the middle line (the accurate median position being, however, dependent to some extent on the state of the teeth). The tongue and mouth should be moist. Its colour should be pink, and the shape of the papillæ visible, the filiform papillæ being each surmounted by a delicate tipping of fur, of a gray or yellowish colour; or this coating may be more general. This fur is a perfectly normal condition, and is always present in adults after the mouth has been at rest for a time, as, for instance, on waking in the morning. In infants, however, it is nearly absent, no doubt because the filiform papillæ are not yet developed. On the nature of this "fur" or "coating" there has been a good deal of discussion; and even now

two eminent authorities, Dr. Dickinson and Mr. Butlin (9), are not quite agreed as to the share of the various elements in its production. The microscope shows that on the surface of the tongue there is always a variable amount of epithelial débris and food remains; but besides this there is also some parasitic growth—of micrococci chiefly, but also of bacillus subtilis—which is specially attached to the summits of the filiform papillæ. Dickinson maintains that the epithelial element predominates; whereas Butlin considers the micro-organisms to be the most important constituent, and that they increase in number as the fur thickens, whereas the epithelial element decreases. Certainly the conditions under which fur increases—tongue-rest, warmth and moisture—coincide with the conditions generally favouring bacterial growth. In certain cases the furring may be confined to one side of the tongue; this is usually due to defective movement on that side, as in hemiplegia; or to a jagged tooth irritating one side of the tongue. Possibly in some cases the phenomenon may depend on unilateral nerve influence.

Dr. Dickinson (15), in his Lumleian Lectures delivered in 1888, has so fully analysed the varieties of fur that I think a short summary of his remarks will be useful. He divides the appearances as follows:—*Stippled or dotted tongue;* an exaggeration of the normal condition, generally due to mere want of use (such as is caused by taking liquid diet, the influence of which on furring is very great), not to pyrexia; present in local or organic disease. *Stippled and coated;* here both the filiform papillæ and the intervals between them are covered with fur. This occurs with increased pyrexia and commencing depression. *Coated and plastered tongues;* in the first there is a continuous layer, in the second a superabundant coat of fur, which is of a dirty white, gray, or yellowish colour: this may thin off at the edges, or break away abruptly. In the plastered tongue the fur, at first white, gradually becomes dirty and brown, and if this state continue it becomes dry. The saliva is deficient. As the fur increases the papillæ become elongated, partly from overgrowth and partly from want of use. In the *strawberry* tongue the same condition exists, but the fungiform papillæ are especially hyperæmic and, being free from fur, stand out prominently. The two latter tongues occur in acute and febrile diseases, such as acute rheumatism, pneumonia, and acute infections; and the last form is seen especially in scarlet fever. *The furred or shaggy tongue;* here the filiform papillæ project markedly, and on their tips, especially in the front of the tongue, there are brown spots; while the central and back parts are covered with irregular pointed masses or brown crusts, which give the whole tongue a shaggy, rough appearance. The tongue is generally dry and the saliva deficient, which results in a hardening of the fur and consequent protection of the papillæ. This is a later stage of the preceding conditions; it signifies prostration is increasing to an extent that makes the prognosis grave. If no improvement takes place it passes into the *incrusted, dry and brown tongue*, where the intervals between the papillæ are filled up, and the tongue appears covered with brown, dry incrustation which is broken and fissured irregularly. At the

same time similar incrustation takes place on the teeth, producing "sordes." The deposit on the tongue fills the intervals between the elongated papillæ, and also covers them with a uniform layer, which may be as much as a quarter of an inch thick: it consists of epithelium and much parasitic growth, which is made up partly of micrococci and partly of oidium albicans, mixed with indefinite débris. The brown colour is chiefly due to dryness, from deficiency of saliva and oral respiration; but is partly the result of staining by swallowed liquids. This appearance is coincident with more severe depression, such as is present in the later period of enteric fever and the comatose state; and it is even of more serious import than the shaggy tongue.

The incrusted tongue may become *red, smooth and dry*, or even actually *bare*. The dry crust, being imperfectly attached to the epithelium underneath, wears off and breaks away, leaving a red, dry surface beneath, covered with a thin translucent membrane which gradually spreads over the whole tongue. This soon becomes fissured by fine lines. At the same time the underlying corium is inflamed, and in places remains actually exposed. This may occur over a considerable extent of surface, and is then apt to be the seat of the growth of such fungi as the oidium. Saliva is here usually absent. Such a tongue occurs in patients who are extremely prostrate, especially where there is much suppuration. The prognosis is extremely unfavourable.

The appearance of the tongue and its varieties of fur must rather be regarded as an index of the general bodily state than as a proof of the existence of any particular disease. For although there are not a few diseases each of which is associated with a peculiar aspect of the tongue, yet the peculiarity is not invariable, and identical appearances may be found in two or more different affections. Hence we are not justified in drawing more than somewhat general conclusions from the coatings of the tongue. As an illustration of this may be mentioned the "strawberry" tongue, which is often looked upon as distinctive of scarlatina; yet it actually occurs, in quite as definite a form, in many cases of pneumonia.

So again there is no definite connection between changes of the tongue and disorders of the stomach and bowels. The most severe stomach affections and the most prolonged constipation may be associated with a perfectly clean tongue. Diarrhœa, on the other hand, rapidly produces marked furring. This is probably dependent on the dehydrating effect of purging, and acts in the same way as does the deficiency of saliva in the febrile state. In fact this dryness of the mouth and the increased body-heat are the most important factors in the tongue-coating of fevers; promoting as they do, particularly when helped by the quiet of the mouth, the growth of the various micro-organisms which form the bulk of the fur. The influence of the nervous system on the secretion of saliva, the mouth-breathing of coma and of nasal obstruction, must not be forgotten.

Nervous affections of the tongue.—These conditions usually occur as mere symptoms of general maladies, and will be described under those

respective heads; but they may be summarised here for the sake of convenience.

The nerve mechanism of the tongue may be affected in the motor, sensory, or gustatory branches.

Motor.—Involuntary spasms of the genio-hyoglossus occasionally occur which result in uncontrollable protrusion of the tongue. This comes on suddenly, started usually by the act of eating or drinking, and is repeated at fairly regular intervals of some minutes. The condition is rare, but is apt to persist for some considerable time, and causes much distress. After some weeks it passes off, either without treatment or under the influence of tonics, galvanism and change of air.

General convulsive movements of the tongue and mouth are sometimes brought on by attempts to speak—an exaggerated form, in fact, of stammering. These occur most often, but by no means always, in hysterical patients. The prognosis is good, but firm treatment is necessary, and prolonged silence (some weeks or months) has sometimes to be enforced, and graduated and methodical speaking exercises always. The general health must also be attended to.

The irregular jerky movements of the tongue in severe cases of chorea, and the spasm of the tongue and jaw in tetanus, need only be mentioned.

Paralysis of the tongue—unilateral or bilateral—not infrequently occurs as a result of serious organic nervous disease, the seat of the lesion being usually central, as in cerebral hæmorrhage, tumours, embolism, progressive muscular atrophy, and bulbar paralysis. The extent and nature of the paralysis entirely depend on the nervous lesion. Unilateral paralysis occasionally results from hysteria, and may then be associated with hemianæsthesia.

Total organic paralysis of the tongue is a most distressing affection, on account of the extreme difficulty in masticating and swallowing food; usually it is rapidly fatal.

The tremors of certain forms of poisoning—such as alcoholic and mercuric—and those of paralysis agitans, spastic paralysis, and general paralysis of the insane, require mention only. As a result of some central nervous affection, and more rarely of disease of the peripheral trophic nerves, atrophy of the tongue may take place, either equal in degree on the two sides, or unilateral.

Sensation.—Hyperæsthesia of the tongue may occur under the same conditions as of any other part of the body; and occasionally neuralgic pain is confined to the tongue. In these cases usually one-half of the tongue only is affected. The cause is generally obscure, but may be extrinsic irritation from carious teeth or necrosed bone. The pain is so severe, and treatment so unsatisfactory, that surgical measures, such as stretching or excision of a portion of the nerve, have been found necessary. In the slighter cases local applications, such as cocaine or counter-irritation, have sufficed.

Local morphia injections have also been tried with success, but are

open to the standing objection of the danger of inducing morphia-habit. Galvanism applied systematically is sometimes useful.

Anæsthesia is rare; when it does occur it is unilateral, and is usually a symptom of hysteria.

Paræsthesias—usually burning sensations—are present sometimes in women at the time of the climacteric, and epileptic auras also may take this form.

The special sensation of taste may be lost through failure in conductivity of either glosso-pharyngeal, gustatory, trigeminal, or chorda tympani nerves. Its degree varies according to the nerve affected, but the fore part of the tongue usually suffers most. The chorda tympani is the nerve most commonly affected.

The loss of taste may also be secondary to loss of smell from various nasal affections; but in this case the qualities of bitterness, saltness, and sweetness can usually be detected; the flavour or bouquet which is dependent on concurrent smelling is however lost. Loss of taste often occurs as an after-symptom of influenza.

Gustatory paræsthesia occurs sometimes in facial paralysis, and is a common symptom in insane patients, particularly of the melancholic type.

But a very short reference is necessary to the following affections of the tongue, which are more especially surgical:—

Hypertrophy of the tongue, or macroglossia, is usually a congenital affection due to an overgrowth of the connective tissue and a dilatation of the lymph spaces; it is, in fact, a local elephantiasis, and not a true hypertrophy at all. It occurs not infrequently in cretins.

Adhesions of the under surface of the tongue and shortness of the frænum (*tongue-tie*) are congenital defects. The latter is vulgarly believed to be a common affection, but is really a rare one. The cause of the inability to suck, which makes the nurse consider the child tongue-tied, is usually to be sought in nasal obstruction, due perhaps to adenoids, or to congenital syphilis.

Various forms of **cysts** occur: *mucous*, on the dorsum of the tongue, from occlusion of some mucous gland and accumulation of the secretion: *hydatid*, rare, but, when present, near the mesial line in the muscular substance; the tumour is tense and the fluid has the usual characters.

Chronic abscess is also rare; its development is slow, the tumour is smooth, rounded, not translucent or painful, and can best be diagnosed by incision.

Ranula is a smooth swelling, usually unilateral, on the under surface of the tongue near the frænum, having a deep bluish tint, translucent and fluctuating. Its course is chronic, and it is due either to dilatation of Wharton's duct or to expansion of the acini of a mucous gland.

Dermoid cysts are much rarer, though they occur in very similar position to ranula. When large, however, they project into the neck more than ranula does. Their contents have the characters usual in such tumours.

Actinomycosis occasionally affects the tongue.

The *innocent* **tumours** found in the tongue are the following, most of them somewhat rare: Lipoma, fibroma, chondroma, osteoma, papilloma, adenoma (very rare), angioma; and the *malignant*, sarcoma (very rare), and carcinoma (already referred to, and of course far more common). Syphilis in its later stages also gives rise to tumour-formations.

The differential diagnosis and treatment of these various affections will be found in the surgical text-books (2, 3, 5).

Affections of the Salivary Glands

Hypersecretion of saliva, or ptyalism, is now a less common condition than it was formerly, when mercurial treatment was not considered complete until salivation had been produced; for mercurial poisoning is certainly the most common cause of ptyalism.

The amount of saliva normally secreted in twenty-four hours may be put at one to two quarts, whereas hypersecretion may amount to even as much as five quarts: but the character of the saliva under these circumstances is much altered; the solid constituents do not increase proportionately, and after the excessive secretion has continued for some time, the specific gravity may fall from 1006 (its normal degree) to 1001 or 1002. Hypersecretion is a most distressing condition, as, if at all severe, the patient is unable to swallow all the saliva, and is obliged to be constantly spitting it out; during sleep it dribbles from his mouth on to the pillow.

With regard to mercurial salivation individuals show a marked idiosyncrasy; some persons tolerate large doses for a long period, whereas in one case under my own notice a single dose of 3 grs. of blue pill set up salivation lasting two days. Infants seem to tolerate proportionately larger doses than adults; this may be due to the slight degree of development of their salivary glands. Other drugs producing salivation are iodide of potassium, arsenic, copper, and pilocarpine; the last named possesses the power, when given hypodermically, of producing immediate and most uncomfortable salivation. Various extrinsic irritations from such organs as the liver, spleen, and genital organs are stated to produce it occasionally, and it sometimes occurs as a symptom of the early stages of pregnancy, and may even persist until delivery (35).

Dentition in infants and ulcerative stomatitis often result in salivation. It is a frequent and distressing symptom in small-pox (19), and the hypersecretion of viscid saliva in hydrophobia is most distracting to the patient.

In bulbar paralysis, and the stuporous form of melancholia occurring in young adults, dribbling of saliva is a very characteristic symptom; but it is questionable in these cases how far it is due to real over-secretion and how far to mere overflow.

Diminished secretion (aptyalism, xerostomia). — Certain persons suffer from a constant insufficiency of saliva, though there be no obvious

disease of the salivary glands, or any ascertainable cause, unless it began as a consequence of fright or shock, or in the course of hysteria. The same symptom occurs in diabetes and in the more severe febrile states. According to Dr. Dickinson (16), the more pronounced degrees of tongue-coating with dryness are chiefly due to the absence of saliva, and not to the drying influence of oral respiration. In proof of this statement Dickinson frequently catheterised the parotid duct to ascertain the amount of saliva secreted, and where the tongue was dry, he invariably found a great diminution in the quantity. He quotes some interesting cases bearing on this point. The influence of the nervous system is well shown by the dry mouth of persons who are in a state of nervous tension under circumstances of danger or anxiety. The "ordeal by rice" of Eastern nations is an instance in point.

The condition may continue—when it occurs independently—for some months or even years, and *treatment* is extremely difficult. Pilocarpine may be given by the mouth ($\frac{1}{15}$ gr. several times daily), or galvanism over the glands may be tried. Frequent oily or moist applications to the tongue afford temporary relief.

Inflammation of the salivary glands.—The salivary glands are liable to inflammation which may be of a specific nature, or metastatic in origin. The specific inflammation is called "Mumps," and will be discussed under that head; but it is as well to mention that though it is called Parotitis the other salivary glands as well as the parotid are usually affected.

In *metastatic parotitis* the inflammation is more strictly confined to the parotid gland. The term "metastatic" is, however, not a good one, as the inflammation is not complementary to that of any other organ, but merely occurs as a symptom or complication of certain other maladies. The list of such diseases is tolerably large. Various fevers—scarlatina, enteric, typhus, and rheumatic; pyæmia, pneumonia, peritonitis, and phthisis; gout (28), ovariotomy and catheterisation; gastric ulcer, especially when rectal feeding is resorted to; dysentery, and intestinal obstruction are the chief of them.

This form of parotitis differs from mumps both by the limitation to the parotid gland—usually, indeed, to one parotid only—and in its course, which is almost always towards suppuration. If an abscess form and be left unopened, the pus is apt either to break into the external auditory meatus, or to burrow somewhat deeply into the pterygoid region or between the fasciæ of the neck. The *treatment*, therefore, consists in early incision when any sign of suppuration appears to exist; but poulticing, belladonna fomentations, ice-bags, or leeches may first be tried. It is a serious complication, but not so serious when occurring late in the disease, or during convalescence, as at any earlier stage.

It seems likely that the origin of this curious condition is to be sought, in some cases at least, in the diminution of the secretion of saliva caused by the absence of buccal stimulus; its frequency in the prolonged rectal feeding, necessary in severe cases of gastric ulcer, is an instance of this

cause. On the other hand, in some cases it is undoubtedly due to the same cause as other pyæmic abscesses.

Among the surgical affections of the salivary glands salivary calculus may be mentioned—a concretion consisting mainly of carbonate and phosphate of lime, and occupying any one of the salivary ducts, but most frequently that of the submaxillary gland—and salivary fistula, an opening in connection with the salivary duct on the surface of the cheek, usually due to some wound or abscess communicating with the duct.

W. A. WILLS.

REFERENCES

1. AUDRY. *Rev. de méd.* 1887. — 2. BAKER, MORRANT. "Diseases of Tongue," Heath's *Dict. of Surgery*, 1886.—3. BARKER. Holmes's *System of Surgery*, vol. ii. 3rd edit. p. 563.—4. *Ibid.* p. 560.—5. BUTLIN. *Diseases of Tongue*, 1885, p. 43.—6. *Ibid.* p. 126.—7. *Ibid.* p. 159.—8. *Ibid.* p. 31.—9. *Ibid.* p. 388.—10. BUTLIN. *Med.-Chir. Trans.* vol. lxi. p. 51.—11. CROCKER, R. *Diseases of the Skin*, 2nd edit. 1893, p. 187. —12. CLARKE, FAIRLIE. *Med.-Chir. Trans.* vol. lvii. p. 155. — 13. DEBOVE. *Le Psoriasis buccal.* Paris, 1873.—14. DICKINSON. "The Tongue as an Indication in Disease," *Lumleian Lectures for* 1888, p. 81.—15. *Ibid.* p. 28.—16. *Ibid.* p. 85.—17. DUNN. Donkin's *Diseases of Childhood*, 1893, p. 28.—18. FAGGE. *Prin. and Pract. of Med.* 2nd edit. vol. ii. p. 300.—19. *Ibid.* 3rd edit. vol. ii. p. 133.—20. FOX, CALCOTT. *Lancet*, 1884, vol. i. p. 842; and *Brit. Jour. of Dermat.* 1889, p. 353.—21. FRAENKEL. *Internat. klin. Rundschau.* Vienna, 1891.—22. GRAHAM. *B.M.J.* vol. i. 1887, p. 61.—23. GUBLER. Art. "Bouche," *Dict. Encyc. des Sc. méd.* vol. x. pt. 1, p. 234.—24. HULKE. *Clin. Soc. Trans.* vol. ii. p. 1. — 25. HUTCHINSON. Lectures R.C.S., *Med. Press and Circ.* vol. ii. 1883, p. 1.—26. JACOBI. *Trans. of the Assoc. of Amer. Phys.* 1894.—27. MANSON. *Med. Reports Imp. Chinese Customs*, 1879-80, Issue xix. p. 33.—28. *Med.-Chir. Trans.* 1887, vol. lxx. p. 217.—29. MYGIND, HOLGER. *Journal of Laryngology*, London, Jan. 1890.—30. NEDOPIL. *Arch. für klin. Chir.* Bd. xx. S. 324; 1877.—31. NESVIZKI. *Uratsch*, No. 15. St. Petersburg, 1891.—32. PARROT. *Med. Times*, 1881, vol. i. p. 461.—33. PAYNE *St. Thomas's Hospital Reports*, 1883.—34. PEPPER'S *System of Medicine*, "Diseases of Mouth."—35. PLAYFAIR. *The Science and Practice of Midwifery*, 4th edit. vol. i. p. 222.—36. ROTH. *Wien. med. Presse*, 1887.—37. SAJOUS'S *Annual of the Univ Med. Sciences*, 1892, vol. i. c. 2.—38. SANSOM. *Med.-Chir. Trans.* vol. lxi. p. 1. — 39. SCHEET. *Münch. med. Woch.* 1887. — 40. SCHWIMMER. *Vierteljahresschr. f. Dermat. und Syph.* N.F. 1877, p. 511. — 41. SEVESTRE. *La semaine méd.* July 1, 1891.—42. STOCKER. *B.M.J.* vol. i. 1884, p. 601.—43. *Ibid.* vol. ii. 1886, p. 629.—44. THIN. *Practitioner*, London, 1883, vol. xxxi. p. 169.—45. *Ibid.* 1887, vol. xxxix. p. 337.—46. THOMPSON, W. H. Sajous's *Annual*, 1888, p. 293.—47. *Westminster Hospital Reports*, vols. i.-ix.—48. VAN DER BURG. *Med. Reports Imp. Chinese Customs*, 1883-84, Issue xxvii. p. 55.

W. A. W.

DISEASES OF THE ŒSOPHAGUS

A BRIEF description of the anatomy of the œsophagus, and a few remarks on the methods of examining the part, will not be out of place as a preface to a consideration of its diseases.

Anatomy.—The œsophagus begins at the lower border of the cricoid cartilages on a level with the sixth cervical vertebra. The commencement of the œsophagus is six inches from the incisor teeth; starting in the middle line it deviates to the left in the neck, but in the thorax returns to the middle line at the level of the fifth dorsal vertebra. Below this it inclines again to the left to pass through the muscular opening in the diaphragm at the level of the tenth dorsal vertebra. It enters the cardiac end of the stomach, opposite the upper border of the body of the eleventh dorsal vertebra, after a short abdominal course of half an inch. The distance from the stomach to the incisor teeth is 15½ inches. The œsophagus proper is 9½ inches long, and is narrowed (i.) at its commencement; (ii.) at a point 2¾ inches lower down, marking the situation of the diverticulum which in fœtal life passes off to form the lungs; and (iii.) where it penetrates the diaphragm.

In the neck the œsophagus is in relation in front with the trachea and the left lobe of the thyroid gland, while the recurrent laryngeal nerves run in the groove between the trachea and the œsophagus. Laterally it is in relation to the lobes of the thyroid gland and the common carotid arteries, especially on the left side. Posteriorly it is only separated from the bodies of the vertebræ by fascia and the longus colli muscles, and is therefore easily displaced by an abscess in connection with caries of the spine.

In the thorax the trachea, left bronchus, bronchial glands, pericardium, and left auricle are in front. Laterally it is in relation with both pleuræ, more especially the right. The plexus gulæ is on its anterior surface, and the left and right vagi respectively in front and behind. The aorta lies to the left of the œsophagus above, and crosses behind it to reach its right side just above the diaphragm. The thoracic duct, for the greater part of its course, lies behind and to the right of the œsophagus; opposite the fourth dorsal vertebra it crosses behind it so as to lie on its left side in the superior mediastinum. Posteriorly the œsophagus lies on the bodies of the vertebræ and the right intercostal arteries.

The surface-markings of the œsophagus may be taken as extending from a point one inch above the vertebra prominens, to a point in the middle line rather below the level of the inferior angles of the scapulæ.

The internal surface is covered with stratified squamous epithelium, and is normally thrown into rugæ. The muscularis mucosæ separates this internal mucous coat from the loose submucous layer. Small

racemose glands are found in the submucosa; they are more numerous in the lower third of the œsophagus. The submucosa, unlike that of the rest of the alimentary canal, does not contain any lymphoid tissue. It has been said that this peculiarity accounts for the comparative immunity of the œsophagus from inflammation.

The outer longitudinal muscular coat is thin, and is in part connected with the inferior constrictor of the pharynx above and the longitudinal muscular coat of the stomach below. The thicker circular coat is also connected with the inferior constrictor; while below it joins the circular and oblique muscular coats of the stomach. The circular coat forms a sphincter for the cardiac orifice of the stomach. The tonic contraction of these fibres must be inhibited before the contents of the stomach can be vomited; ineffectual retching has been attributed to failure of this relaxation. Sometimes complete incompetence of the cardiac sphincter follows operations on the stomach, such as gastro-enterostomy; so that food runs back into the œsophagus after being swallowed (9).

The lower half of the muscular coats of the œsophagus is composed of the smooth involuntary variety; the upper half contains striated muscular fibres which in the middle third become mixed with smooth fibres.

Externally there is a layer of areolar tissue.

The sensory nerve fibres are derived from the superior laryngeal and pharyngeal branches of the vagus; the exterior laryngeal supplies the upper part of the œsophagus with motor power; the lower part is supplied from the plexus gulæ.

The arteries are derived from the aorta, and anastomose with œsophageal branches of the inferior thyroid and coronaria ventriculi, and run in a longitudinal direction.

The veins open into the azygos veins; the anastomoses between them and the gastric veins may become greatly developed in cases of hepatic cirrhosis, and by rupturing may give rise to fatal hæmatemesis.

The lymphatics form a network in the submucous layer, and open into the posterior mediastinal glands.

Methods of examining the œsophagus.—By external examination enlarged cervical glands, secondary to carcinoma of the œsophagus, may be felt; and their discovery would help to clear up any doubt as to the cause of dysphagia. A pharyngeal pouch may be felt to swell up when food is taken, and can be emptied by pressure applied externally.

Internal examination gives information as to the presence and situation of a stricture or foreign body.

Œsophagoscopes and endoscopes have been described, but are hardly ever used in practice.

The two ordinary means at our disposal are:—

(i.) *The passage of an œsophageal tube or bougie.*—The pharynx may first with advantage be sprayed with a 10 p.c. solution of cocaine. The bougie should be warmed and lubricated with glycerine, not with oil, which is very repugnant to the patient. The patient sits in a chair and

the bougie is passed along the posterior wall of the pharynx into the œsophagus. The bougie is very likely to be arrested as it passes into the œsophagus behind the cricoid cartilage, about six inches distant from the incisor teeth, without there being any morbid condition present. The patient should then be directed to swallow, in order to help the onward passage of the instrument.

The point at which any obstruction is encountered should be noted by measuring the distance from the incisor teeth. The distance from the incisor teeth to the stomach is at least 15½ inches, and may be even more.

By means of the bougie, spasm, stricture due to organic causes, and paralysis can be distinguished from each other. If, in the case of stricture, a bougie with a small depression near the end is passed, some of the growth, if it be a case of carcinoma, may be removed and examined microscopically and the diagnosis settled (4).

It has been suggested (16) that a binaural stethoscope should be connected with a perforated œsophageal tube, and that in this way friction due to an early stricture may be detected. A stricture thus discovered in the early stage could be more satisfactorily treated by dilatation than in a later stage.

(ii.) *Auscultation* of the œsophagus was first practised by Hamburger, and was described in this country by Mackenzie and Professor Clifford Allbutt. The œsophagus is auscultated from without while the patient swallows water or gruel. In order to employ this method the observer must make himself familiar with the characters of the normal œsophageal sound. The rapidity, or rate at which the morsel is swallowed, can be timed by placing the hand on the hyoid bone.

In organic stricture the sound of the passage of the morsel is much altered, prolonged, or transformed into a continuous and confused noise; in spasmodic stricture the sound is unaltered; in paralysis of the gullet it is absent.

Skiagraphs taken by means of the X Röntgen rays have already been useful in demonstrating the presence and position in the œsophagus of foreign bodies, such as coins. It remains to be seen whether, on further elaboration, this method will be capable of showing the presence of new growths and of other morbid conditions, such as dilatation.

MALFORMATION.—*Congenital malformations.*—Congenital atresia. In this rare condition the œsophagus is divided into two parts. The upper segment, continuous with the pharynx, ends blindly a short way above the level of the bifurcation of the trachea. The lower segment opens in a normal manner into the stomach, but takes its origin from the trachea above the bifurcation. At first sight this condition might be regarded as analogous to that of imperforate anus, and to be due to a failure of union between the stomodæum and the foregut. But Mr. Shattock has shown that it is in reality due to a secondary obliteration of the œsophagus occurring opposite the diverticulum from which the lower part of the trachea and the lungs are developed. This explanation

is supported by the fact that the pharynx is derived, not from the buccal invagination of the epiblast (stomodæum), but from the foregut. Infants presenting this malformation are of course unable to swallow, and the amount of fluid vomited is the same as that taken. They die of starvation about the fifth day after birth.

More extreme degrees of malformation have been met with in monsters.

The *persistence of branchial clefts* may lead to a fistulous communication between the pharynx and the exterior. The external opening is situated along the anterior border of the sterno-mastoid muscle, and is often associated with the presence of an accessory auricle. Either the internal or the external orifice may be closed. If both are obliterated the intervening portion may undergo cystic dilatation; the contents of these branchial cysts are either mucoid or dermoid in character. The multilocular serous cysts of the neck (Hygroma) are not derived from the branchial clefts.

In rare instances carcinoma may arise in these branchial cysts.

The branchial fistulæ or pouches are, it is to be noted, lateral in position, and communicate with the pharynx, not with the œsophagus.

Acquired malformations—Pressure pouches.—Syn.: Œsophageal, pharyngeal pouches, pharyngoceles, pulsion diverticula. Pressure pouches, though often called œsophageal, in reality arise from the lower part of the pharynx, just at its junction with the œsophagus. This is, however, a convenient place to describe them.

A pressure pouch begins in the posterior median line as a small depression which gradually becomes larger from the lodgment of food in it. A diverticulum is thus formed between the œsophagus and the spine, which points downwards; and, from the inclination of the œsophagus to the left, often projects on that side of the neck; though it may present on both sides. The orifice appears as a transverse slit. As the pouch gets larger more and more food enters it, and the pressure thus exerted on the œsophagus renders swallowing more and more difficult. Structurally they are composed of thickened mucous membrane covered by connective tissue. The neck of the sac is enclosed by muscular fibres derived from the inferior constrictor muscle; but although the pouches look as if they had a muscular wall, microscopic examination often shows that this is not the case. Their wall, however, is as thick as that of the œsophagus.

These pressure pouches vary very considerably in size; they may contain as much as six ounces. They are elastic and can be distended to twice or three times their ordinary size. Their average length is four to five inches. Dr. Newton Pitt has recorded a case in which squamous-celled carcinoma developed in a pressure pouch. There are two views as to their causation:—

(i.) That the pouches are congenital in origin and become gradually dilated. As has been already seen, the diverticula resulting from partial persistence of branchial clefts are lateral in position; while these pressure

pouches are situated in the posterior median line. It is highly improbable, therefore, that these two conditions have anything to do with each other. The fact that the symptoms are first noticed past middle life, or even later, is also against the view that they are congenital, or in any way analogous to Meckel's diverticulum in the ileum.

(ii.) That as a result of traumatism, applied either from without or from within, a hernia of the mucous through the muscular coat occurs.

The constant situation in which those pressure pouches or pharyngoceles occur depends on the anatomical structure and arrangement existing at the junction of the pharynx and the œsophagus. The lumen of the passage narrows considerably in this situation. The anterior wall being formed by the cricoid cartilage is firm and unyielding, while the posterior muscular wall at this point is thinner and weaker than in any other part of the pharynx or œsophagus. This condition is, of course, congenital; but there is nothing to suggest that the pouches themselves are congenital in origin.

A hard bolus of food being driven against this place of less resistance might produce a hernial protrusion of the mucous membrane which, as time went on, would become progressively larger. Thus traumatism may be the immediate starting-point of such a pouch; but the situation is determined by the normal anatomy of the part.

Stricture of the upper part of the œsophagus and pressure exerted from without by tumours, such as goitres, have been supposed to act as causal agents.

Pressure pouches are rare, but when they do occur they are met with most often in patients past middle life or even in old age.

Though symptoms are not known to have been present in all the specimens, they have been well marked in most of the recorded cases. The usual course of the disease is as follows:—

First, there is return of undigested food some time after a meal, perhaps with coughing. This regurgitation increases in amount, and is after a time associated with distinct difficulty in swallowing. As the pouch increases in size the swelling of the neck after eating or drinking becomes more noticeable. The pouch can be emptied by pressure applied from without. Swallowing gradually becomes very difficult, and a probang which previously could be passed down the œsophagus now enters the pouch. Finally, unless relieved, the patient dies of slow starvation.

Treatment.—Removal of the pouch, as described by Von Bergmann and Mr. Butlin (5), has given brilliant results; and is the radical and ideal treatment. Life has been prolonged by gastrostomy; but in these cases the cause of the dysphagia was not correctly diagnosed till after death.

Traction diverticula generally occur on the anterior wall of the œsophagus near the bifurcation of the trachea, and are commonly due to adhesions between inflamed bronchial glands and the wall of the œsophagus. As the glands and adhesions contract, the wall of the œso-

phagus at this spot is pulled out, and a pouch results. Traction diverticula may occur elsewhere; thus I have seen one on the postero-lateral aspect due to the contraction of adhesions between the right lung and the œsophagus. They are never more than an inch deep.

They give rise to no symptoms unless ulceration of the wall of the diverticulum occur. This may lead to a tracheo-œsophageal or broncho-œsophageal fistula, and so to septic broncho-pneumonia and gangrene of the lung. In a similar way, pleurisy, pericarditis, or a mediastinal abscess may be set up.

Dilatation of the œsophagus.—Contrary to what might have been expected, and indeed is often stated, dilatation above a stricture of the œsophagus is far from common. In most cases food, if unsuccessful in passing through the stricture into the stomach, does not accumulate to any extent above the obstruction, but is rejected shortly after being taken.

When dilatation secondary to a stricture does occur, the point of obstruction is usually near the diaphragm. Dilatation may follow chronic œsophagitis.

In *primary dilatation*, wherein there is no manifest mechanical cause to account for the condition, the whole length of the œsophagus is affected; but the middle third of the tube is most dilated, so that the œsophagus has a spindle-shaped outline tapering towards its two ends. At the point where it passes through the diaphragm the œsophagus is usually of its normal size and presents no sign of organic stricture.

The dilated œsophagus may become lengthened, and by bending on itself present a tortuous outline. The muscular walls are usually hypertrophied, and the mucous membrane may be normal, thickened, inflamed, or ulcerated.

Etiology.—The cause of this somewhat rare condition—primary dilatation—is not satisfactorily known. Morell Mackenzie considered that it was due to atony, either congenital or acquired, or to weakness of the œsophageal wall on its whole circumference; and the latter is the generally accepted view.

It is remarkable that the muscular coat of the œsophagus should become hypertrophied in cases of simple dilatation. Dilatation with wasting and thinning would be a natural result of muscular weakening; but dilatation with hypertrophy forcibly suggests some obstruction at the cardiac end, and if no organic stenosis be present it is possible that some functional obstruction existed during life. This could be explained by continued closure of the cardiac orifice of the œsophagus, which might conceivably be due to a spasm of the sphincter fibres at the lower end of the circular coat of the œsophagus; or to paralysis of the longitudinal muscular fibres, by the contraction of which the cardiac orifice of the stomach is opened.

Continued spasm might be expected to lead to marked local hypertrophy of the circular muscular coat near the cardia; such a condition, however, does not seem to have been observed in the cases recorded.

By the alternative view paralysis of the longitudinal muscle would explain dilatation, by leaving the cardiac orifice closed it would lead to obstruction at the lower end of the œsophagus, and thus might induce general hypertrophy in the circular coat. Simple dilatation with hypertrophy, then, can perhaps be explained more satisfactorily as the result of inhibition and atony of the external longitudinal coat than as due to weakness of the whole of the muscular wall; but the explanation cannot be said to be in any way proved. We can only say that dilatation of the œsophagus, like the same condition in other hollow muscles—such as the heart, bladder, stomach, intestines—is usually accompanied by hypertrophy; but the hypertrophy, especially in the thinner-walled viscera, usually fails to compensate or to correct the dilatation.

Symptoms.—Regurgitation of food some hours after eating in an undigested state, except for some transformation of starch into sugar. The breath is usually foul and there is often salivation. Pain of a burning character may be felt in the gullet. The disease is slow in progress and need not necessarily shorten life.

Treatment is palliative, and consists in the administration of small quantities of fluid food at frequent intervals. The treatment of dilatation secondary to stricture is of course that of the primary affection.

ŒSOPHAGITIS.—**Thrush** or parasitic stomatitis, due to a fungus usually spoken of as *Oidium albicans*, but perhaps more correctly described as *Monilia candida*, may spread from the mouth and pharynx of infants to the œsophagus. It may attack the whole length of the gullet or but a part of it; the lower half of the tube near the stomach is more often affected. It may be discrete or confluent, and by exuberant growth may actually occlude the lumen, and so interfere with swallowing.

There may be no symptoms to suggest that the œsophagus is affected; or, on the other hand, swallowing may be difficult or even impossible.

Diagnosis.—Its presence may be suspected when the above symptoms arise in a case of thrush in the mouth; but otherwise it can hardly be recognised.

The *treatment* is much the same as that of thrush in the mouth; namely, attention to cleanliness in feeding and the local action of borax or similar preparations given by the mouth.

Thrush may also occur in adults in the last stages of wasting diseases such as phthisis.

Diphtheria.—In very rare cases diphtheritic inflammation may spread from the fauces into the œsophagus; and membrane has been found to extend even into the stomach. It gives rise, however, to no characteristic symptoms, apart from the fact that the cast of the tube may be brought up.

A pseudo-membranous œsophagitis is very occasionally seen in the course of various febrile diseases.

Simple acute œsophagitis.—*Acute catarrhal œsophagitis.*—Simple

acute inflammation may attack the œsophagus; but it is a disease that is rarely seen, and its etiology is equally obscure. It may spread from the pharynx, and has been attributed to the local action of alcoholic drinks, to hot food, to cold applied either internally, as in eating ices or drinking iced water, or externally. It has also been attributed to rheumatism.

It is said to occur sometimes in the course of the specific fevers, and to account in some degree for the anorexia met with in them.

Extension of inflammation from adjacent parts—for example, in pericarditis, pneumonia, or pleurisy—is seldom manifested clinically by pain or difficulty in swallowing. Post-mortem examination, however, often shows that the inflammation has spread at any rate to the muscular walls of the œsophagus.

Lastly, acute œsophagitis may arise without any assignable cause, especially in sucklings.

Phlegmonous inflammation of the œsophagus is a rare and very severe affection. It may follow the acute catarrhal form or the impaction of foreign bodies. The spread of suppuration from without, or from an ulcer in the wall of the tube, may give rise to it. The mucous coat may be separated or dissected off by diffuse suppuration occurring in the submucous coat. Ulceration or even gangrene (gangrenous œsophagitis) may result. It may rapidly prove fatal in a few days; if recovery take place, cicatricial contraction is likely to follow and to lead to stricture.

Symptoms.—In severe cases there is extreme pain on swallowing even liquid food, so much so that the patient hardly dare try to relieve the urgent thirst which characterises the affection, or to swallow his saliva. When an attempt is made there is very considerable difficulty due either to reflex spasm or paralysis. Speaking and movement are painful, there is tenderness on pressure, and complaint may be made of a dull aching pain in the neck. There is expectoration of a glairy mucus which, in cases where suppuration occurs in the walls of the œsophagus (phlegmonous œsophagitis), may contain pus and blood.

Treatment in severe cases consists in absolute rest to the œsophagus, the patient being fed by enema. Pain and discomfort may be relieved by morphia and by the external application of poultices and hot fomentations. In milder forms demulcent drinks and ice may be given by the mouth, the patient should be put on a low diet, and any concomitant pharyngitis or gastric disturbance treated.

Traumatic œsophagitis.—Acute traumatic œsophagitis may be due to swallowing very hot fluids—such as boiling water, or inhaling steam from the spout of a kettle. Children are especially apt to injure themselves in this way. But most frequently it is the result of irritant or corrosive poisons taken by accident, or with suicidal intent. Strong solutions of acids and alkalies produce necrosis of the mucous membrane, which may be expelled as a complete cast; and occasionally gangrenous œsophagitis may result.

Weaker solutions give rise to varying degrees of inflammation and

ulceration. The mucous membrane of the œsophagus may be affected in parts only, and is often much less damaged than that of the stomach.

Symptoms.—Immediately on taking the poison there is a burning pain in the gullet extending to the stomach, and great pain on attempting to swallow. The symptoms may be somewhat masked at first by collapse; but as the patient rallies he becomes characteristically anxious. Glairy mucus stained with blood may be brought up. The lips, tongue and mouth will show the effects of the poison; unless, as sometimes happens in suicides, the poison were carefully poured by means of a long-necked bottle into the pharynx.

The dysphagia and pain continue, and the symptoms resemble those of acute catarrhal œsophagitis; though they are of course more marked in degree.

Treatment.—If the poison taken was an acid, it should be counteracted by dilute alkaline solutions, which may be given by a small tube passed by way of the nose half-way down the œsophagus, but not by the stomach-pump. Injury by alkalies should be treated, not by the administration of mineral acids, but of dilute vinegar or oil.

Liquid food only should be given by the mouth, and the patient's strength maintained by enema.

After-results.—In severe cases the difficulty of swallowing, due to the inflammation and ulceration, passes uninterruptedly into that due to the resulting cicatricial contraction. In milder cases there is an interval between the initial dysphagia and that due to cicatricial stenosis; stricture usually makes itself manifest within a year after the occurrence of traumatic œsophagitis.

Chronic œsophagitis.—Chronic inflammation of the œsophagus may accompany other diseases of the tube, such as cancer or stricture; and may be associated with syphilis or phthisis (Mackenzie); but it is rarely seen post-mortem, and, generally speaking, is not recognised clinically. It is probable that in many cases of chronic gastritis, especially when there is pharyngitis as well, there is also some chronic œsophagitis; but the manifestations of the former overshadow any that may be due to changes in the œsophagus. The relationship between chronic alcoholism and chronic œsophagitis is doubtful.

In chronic heart disease, and in other cases where the œsophagus has been exposed to external pressure—as, for example, from a mediastinal growth—chronic œsophagitis may be met with.

Morbid anatomy.—There is a general thickening and opacity of the mucous membrane, which is covered by tenacious mucus. In places there are small elevations or warts composed of thickened epithelium covering hypertrophied papillæ; the submucosa is unaffected. These warts are of little clinical importance.

In psilosis or sprue, the œsophagus (24) is denuded of epithelium, and shows extensive inflammatory changes on the underlying coats. There is considerable pain on swallowing, as might be expected. The

chronic œsophagitis here is part of the disease, and can be recognised [*vide* article on "Sprue" in this volume].

Chronic œsophagitis may, it is thought, lead to ulceration, and thus by perforation to periœsophageal abscess; or it may dispose to rupture. Cicatrisation of such ulcers may explain some cases of œsophageal stricture of otherwise obscure origin.

STRICTURE OF THE ŒSOPHAGUS.—The œsophagus may be pressed upon from without; thus, in the neck, tumours in connection with the thyroid body may lead to dysphagia; while in the thorax a mediastinal new growth, dislocation backwards of the sternal end of the clavicle, abscess in connection with Potts' disease of the spine, aneurysm of the aorta, or, very rarely, a large pericardial effusion, may compress and narrow the lumen of the œsophagus. But it is remarkable how comparatively tolerant the œsophagus is of pressure exerted from without.

By the word stricture, however, is now meant a narrowing due to some change actually in the walls of the œsophagus; under this heading are:—

(i.) Spasmodic stricture; described under neuroses of the œsophagus (p. 378).

(ii.) Simple stricture: this is a rare condition, and its etiology is somewhat doubtful. When it occurs the stricture is found in one of two situations: (*a*) near the stomach, or (*b*) some little distance below the cricoid cartilage. The stricture shows no evidence of former inflammation, and there is no formation of cicatricial tissue. The stricture is membranous, and consists of a fold of mucous membrane projecting inwards towards the middle of the lumen of the tube, like a diaphragm. It has been thought to be congenital; and very possibly some of the cases occurring in the upper part of the œsophagus are thus explained, and are due to an arrested stage of the kinking process which, when fully developed, gives rise to congenital atresia of the œsophagus.

Against this view, however, is the fact that the symptoms in most cases are manifested in middle or even in advanced life, and were not marked at an early age. Mr. W. Kendal Franks suggests that such a stricture is the result of spasmodic contraction of the muscularis mucosæ, or circular muscular coat; possibly reflex in origin. A few undoubted cases of congenital stenosis of the lower end of the œsophagus are on record.

Fibrous or cicatricial stricture of the œsophagus.—The healing of an ulcer may, by subsequent contraction, lead to progressive and permanent narrowing of the lumen—a process analogous to the production of a urethral stricture. Thus it may follow ulceration set up by the impaction of foreign bodies, or by rough or sharp pieces of food. A gastric ulcer in a somewhat unusual position round the cardiac orifice may lead to œsophageal obstruction. In syphilis the contraction of cicatrices, or the presence of a gumma on the wall of the tube, might give rise to dysphagia. It has been thought that chronic œsophagitis may,

by ulceration and subsequent organisation, account for some cases of fibrous stricture.

But the commonest cause of fibrous stricture is undoubtedly inflammation, ulceration, and subsequent cicatrisation of the mucous and underlying coats, due to the action of acids, alkalies, hot fluids, and the like (traumatic œsophagitis), whether taken accidentally or with suicidal intent.

When a strong solution of a corrosive poison is taken, the primary difficulty (dysphagia) and pain (odynphagia) in swallowing pass almost uninterruptedly into the dysphagia caused by the cicatricial narrowing of the lumen of the gullet. When a weak solution has been taken, the pain and difficulty in swallowing due to the ulceration pass away as the surface heals; but in a few months dysphagia recurs, and, as the cicatrisation progresses, becomes more marked.

The stricture is usually of considerable extent, 2 to 3 inches or more; or there may be several strictures. Above the obstruction the muscular walls may be hypertrophied, and occasionally the tube may be dilated.

Inflammation around the stricture may give rise to a periœsophageal abscess; or the perforation of an ulcer to pleurisy, gangrene of the lung, or an abscess in the immediate neighbourhood of the gullet.

Symptoms.—The dysphagia begins gradually, and is progressive and permanent. Inability to take solid food may come on suddenly, and is succeeded by dysphagia for liquids. From added spasm the obstruction may vary in degree, but never entirely disappears as it does in pure spasmodic stricture. A bougie is always arrested at the same point in its progress; and by means of auscultation the situation of the stricture can be confirmed. Food is returned shortly after being taken, and is alkaline in reaction. The food generally seems to the patient to stop at or about the episternal notch. Emaciation is a natural result of the chronic starvation.

Diagnosis.—The history that an irritant or corrosive poison has been swallowed is an important factor. In cases of syphilis other signs of the disease, and the relief of the symptoms after antisyphilitic treatment, point to a stenosis of specific origin; but the diagnosis of syphilitic stricture can never amount to more than a conjecture (Mackenzie). When there is no history of an injury, an aneurysm should always be first suspected; then carcinoma, and, lastly, simple stricture. If there be the least suspicion of an aneurysm a bougie must not be used; and in malignant stricture great care should be taken, as perforation may be easily produced.

The early symptoms of traumatic and malignant stricture are indistinguishable; but the history of traumatic œsophagitis, and the slow development of the dysphagia, distinguish the former from the stenosis due to carcinoma. Signs of pressure on the recurrent laryngeal nerves should suggest aneurysm or malignant stricture; pressure on these nerves, in innocent strictures of the gullet, is rare.

It should be remembered, however, that carcinoma may arise in a fibrous stricture of some standing.

Several methods of treatment have been advocated and practised; gradual and rapid dilatation, internal œsophagotomy, opening the œsophagus below the stricture or external œsophagotomy, the introduction of a Symonds tube, and gastrostomy.

Rapid dilatation and internal and external œsophagotomy are uncertain in their effects and dangerous in application, and should not be employed.

Gradual dilatation should be first employed, œsophageal bougies of increasing size being passed every other day or so. The bougie is left for some minutes in the stricture, and then withdrawn; the same sized bougie should be introduced several times before proceeding to the next size. When dilatation is complete a bougie should still be passed at intervals of a month, to prevent the occurrence of any relapse. Electrolysis has been employed with success in some cases.

In cases where the diagnosis between cicatricial and malignant stenosis is doubtful, a Symonds tube should be worn (*vide* Malignant Stricture, *infra*). If the stricture have become impassable, and gradual dilatation or the introduction of a Symonds tube be therefore impossible, gastrostomy must be performed without delay. Life may be sustained for some time by rectal feeding, and for this purpose Leube's solid enema is the best; but the operation of gastrostomy should not be postponed until the patient is in extremis (*vide* treatment of Malignant Stricture, p. 372).

In the rare condition of simple or membranous stricture Mr. Kendal Franks has performed œsophagectomy with success.

Prognosis.—If the patient come under observation early, so that the stricture can be treated and his nutrition kept up by food, the outlook is good. If, on the other hand, the narrowing has become extreme and the patient is emaciated, there is danger that he may die of exhaustion before treatment—usually gastrostomy—has had time to work any good.

This form of stricture is compatible, under treatment, with a very considerable lease of life.

Malignant stricture of the œsophagus.—About five cases of sarcoma of the œsophagus have been recorded. In the cases of Mr. Targett (19) and Dr. C. Ogle the tumours were pedunculated. Sarcoma of the lymphatic glands in the immediate neighbourhood of the œsophagus may lead to stenosis; but this is not primary sarcoma of the gullet, and is of course a much commoner event.

The course and symptoms of sarcoma do not appear to differ from those of carcinoma of the œsophagus. Histologically sarcoma of the œsophagus may be a mixed, round, or spindle-celled sarcoma.

Etiology.—Carcinoma of the œsophagus occurs much more commonly in men than in women. In 55 cases, collected by Dr. A. H. Wilson from the post-mortem books of St. George's Hospital, 8 were in women—a proportion of 14·5 per cent; while in 59 cases, collected from various published sources by Mr. Butlin (3), 12 cases occurred

in women—a proportion of 20 per cent. In 100 cases, collected by Mackenzie, 29 women were affected. In 510 cases, gathered together by Mr. Newman, 402 were in men and 108 in women.

In 7297 cases of primary cancer of various parts of the body, collected from the records of four large metropolitan hospitals by Mr. Roger Williams, 2669 were in men; and of these, 144 were primary in the œsophagus—a percentage of 5·3: while in the 4628 cases of cancer in women the œsophagus was primarily affected in 35, or 0·7 per cent.

A family history of malignant disease has been supposed to indicate a proclivity to carcinoma of the œsophagus. Mackenzie found such a history 11 times in 60 cases, and quotes 10 cases, recorded by Richardson, in all of which it was present. In the 55 cases from St. George's Hospital a family history of malignant disease is mentioned in four only; one being included in which a brother of the patient died in the hospital of carcinoma.

Alcoholism and long-continued dyspepsia have been suggested as causes of carcinoma; but there seems little to support this opinion. Voelcker has suggested that enlarged bronchial glands may by irritation be a causal factor in the development of carcinoma of the gullet opposite the bifurcation of the trachea. It is certainly remarkable that carcinoma frequently occurs in the parts of the œsophagus which are normally narrower than the rest, and thus more subject to irritation.

Influence of age.—The disease is rare before forty, and commonest between fifty and sixty. Dr. V. D. Harris has recorded a very exceptional case of carcinoma of the œsophagus occurring in a man aged twenty-one.

	30-40	40-50	50-60	60-70	70-80	80	
Men .	...	8	20	16	3	...	Cases from St. George's.
Women .	...	5	3	...	...	...	
Men .	6	9	22	10	1	1	Butlin's cases.
Women .	2	4	2	1	...	...	

Women are attacked at a much earlier age than men: thus, in 100 cases collected by Mackenzie, all those under the age of forty—eight in number—were in women. The average age in woman is about forty-five; in man it is about fifty-five.

Situation of the growth.—Considerable discussion has taken place as to the commonest site for carcinoma in the œsophagus; the upper, middle, and lower thirds being each thought by various observers to be the part most often attacked.

Mr. Butlin (3) points out that if the œsophagus be divided into halves instead of into thirds, the number of times each half is affected is much the same. This is fairly borne out by the cases from the post-mortem records of St. George's Hospital, as follows:—

Upper third . .	13 (6 of which were in women).
Middle „ . . .	8
Lower „ . . .	14
Upper half . . .	8 (1 in a woman).
Lower „ . . .	12 (1 in a woman).

In 445 cases collected by Newman, the upper third was, however, more often affected than the other two put together; his figures are :—

Upper third . . .	227 = 51 per cent.
Middle „ . . .	98 = 22 „
Lower „ . . .	120 = 27 „

As this author points out, though necropsy reveals the extent, it does not necessarily show the point where the growth began; for it may spread considerably during the patient's life.

When the growth is at the junction of the œsophagus and pharynx there may be some difficulty in determining where it began. This fact, together with the somewhat arbitrary line of demarcation between these continuous channels, may, as Mackenzie points out, account for some of the marked differences in the statistics.

In the cases collected by Dr. Wilson from the records of St. George's Hospital, it is noteworthy that six out of the eight cases of carcinoma in women occurred in the upper third of the tube.

The growth may spread either from the œsophagus to the pharynx or conversely. It is well known that at the lower end of the œsophagus carcinoma may spread to the cardiac orifice of the stomach; and conversely, in two cases of extensive carcinoma of the cardiac end of the stomach I have seen the growth spread up into the submucosa of the œsophagus. The microscopic characters of the growths were incompatible with the view that the growth had spread downwards from the œsophagus. Carcinoma of the cardiac orifice, on the other hand, apart from carcinoma of the cardiac end of the stomach, is primary; not in this organ, but in the œsophagus.

Morbid anatomy.—Carcinoma beginning in the epithelial lining passes into the loose submucosa, and tends to spread transversely round the lumen of the tube; while at the same time it infiltrates the muscular coats. The extent to which it involves the circumference of the œsophagus varies; but frequently, as in other parts of the alimentary canal, carcinoma produces an annular stricture. Obstruction depends chiefly on this annular character of the growth, but also on the projection of the growth into the lumen of the tube. At some distance from the growth small white nodules of a similar structure may occur in the mucous membrane, which Mr. Butlin (3) regards as outlying processes of the main growth, and not as secondary or independent growths. Mr. Targett (20) has recorded a case in which a fibrous polypus of the œsophagus, lying four inches above

a carcinomous growth in the œsophagus, became invaded superficially by the growth; possibly by implantation affected by catheterisation.

Histologically squamous-celled carcinoma or epithelioma is found in an overwhelming majority of the cases; in a small percentage the growth is a spheroidal-celled carcinoma: in this case it is usually scirrhous, but is occasionally medullary. The course of scirrhous spheroidal-celled carcinoma is very slow, and may be twice that of squamous-celled carcinoma; while the medullary form is rapid. Two very exceptional cases of colloid carcinoma have been recorded; and Mr. Newman describes a unique case of columnar-celled carcinoma occupying the lower three inches of the œsophagus: it does not appear that the growth spread from the stomach. Spheroidal-celled carcinoma may be derived from the mucous glands, which are commoner in the lower third of the œsophagus. In Mr. Butlin's fifty-nine cases, fifty-four were of the squamous-celled variety and four of the spheroidal-celled type; the other was Dr. Bristowe's case of colloid carcinoma.

Ulceration begins early; it was absent in three cases only of fifty-five collected from the records of St. George's Hospital. Ulceration of the growth diminishes the obstruction; and, if very early or rapid, may explain the occasional latent cases of œsophageal carcinoma, where no dysphagia is present. Ulcerated particles of growth may in rare cases be vomited up, in which cases microscopic examination of the tissue will settle any doubt that may exist as to the nature of the stricture.

By extension and ulceration the growth tends to invade neighbouring organs. Thus it may so constrict the trachea or bronchi as to lead to suffocation; it may grow by continuity into the lobes of the thyroid body; into the connective tissue of the mediastinum, or into the bodies of the vertebræ. Ulceration may put the œsophagus into communication with the trachea or bronchi, and give rise to a fatal septic broncho-pneumonia; it may perforate into the pleura or lung—usually the right—and set up pleurisy or gangrenous pneumonia. The pericardium is not often put into communication with the œsophagus.

In rare cases ulceration of œsophageal carcinoma may be followed by a localised abscess in immediate relation with the growth.

Ulceration into the aorta or its branches is very rare. Dr. F. Taylor was not able to collect more than nine examples of this accident. When it occurs, death may result from sudden and profuse hæmatemesis.

The recurrent laryngeal nerves are often implicated in the growth; and occasionally the sympathetic is similarly affected.

Secondary growths occur most frequently in the adjacent lymphatic glands, which parts were infiltrated in twenty-four out of fifty-five cases; the liver was attacked in ten cases, the kidneys in five, the bones in four, the suprarenals and the spleen in two cases each. Secondary growths were found six times in the lungs; but in three of these there was a perforation into the trachea or bronchi; so that the growths may have been due to implantation. In one case where the right bronchus was perforated the corresponding lung only was affected.

Symptoms.—These in malignant stricture resemble, in a general way, those of traumatic stricture. Difficulty in swallowing, first of solids and later of liquids, usually comes on gradually; but it may be quite sudden in its appearing. There may be constant dull pain in the gullet, made worse by any attempt to swallow; or severe pain may be felt between the shoulders. But on the other hand the dysphagia is often painless, especially when the growth is in the lower part of the œsophagus (Newman). Return of food shortly after it has been taken is frequent; the vomit may contain frothy mucus stained with blood, or even sloughing portions of the growth. Occasionally swallowing becomes easier after separation of projections of the growth which have blocked up the lumen of the tube. On the other hand, the obstruction may suddenly become absolute from impaction of food in the stricture. As a rule the dysphagia is steadily progressive. Cough is a common consequence; and when there is a communication with the trachea or bronchi it is very severe and is constantly set up by taking food.

Hoarseness, enfeeblement, and even loss of voice may result from pressure on the recurrent laryngeal nerves, and is an indication of malignant rather than of cicatricial stricture. Hunger in the early stages may be a pressing symptom; but, as the disease progresses, the patient becomes indifferent or even unwilling to take nourishment. The breath becomes offensive. Hiccough may be present, and thirst associated with dryness of the mouth may be a troublesome and severe feature. Rapid emaciation, marked loss of weight, and extreme debility are prominent symptoms.

The progress of the disease is rapid; and death from exhaustion, septic absorption, starvation, or from pulmonary complications, occurs within a year from the first symptoms, often much sooner.

Diagnosis.—The history and rapid progress of the disease will usually distinguish it from traumatic, or from simple fibrous stricture; though in the earlier stages, especially if there be no history of the taking of poison, cicatricial stricture may very closely resemble it. Paralysis of one or both vocal cords, or the presence of palpably enlarged glands, point strongly to malignant stricture.

From chronic œsophagitis, spasmodic stricture and paralysis, the special features of each and the cautious passage of a bougie will distinguish it. In chronic œsophagitis and paralysis the bougie will pass without any difficulty; but in spasmodic stricture the bougie will be arrested in different situations, and the obstruction can be overcome by persisting in the pressure.

Pain on swallowing, due to inflammation or ulceration about the larynx, may suggest malignant stricture, especially in tuberculous laryngitis when at the same time there is considerable wasting. The passage of a bougie, however, will show that there is no organic obstruction.

Occasionally the question as to which of the three conditions—mediastinal growth, aneurysm, or carcinoma gulæ—is present, requires

most careful consideration of the physical signs and symptoms; and it may be very difficult to come to a definite diagnosis.

Treatment.—In a few cases where the growth was in the neck the affected segment has been excised; but in the majority of cases it is out of reach. Opening the œsophagus below the stricture (œsophagotomy) is rarely practicable, is much more difficult, and is less certain than gastrostomy. Practically the treatment is palliative only, and consists in careful feeding and the constant wearing of a Symonds tube, or in gastrostomy.

In the earlier stages of malignant stricture Mr. Symonds advocates intubation of the stricture. A short tube, with a projecting rim at the top, which rests on the upper margin of the stricture, is introduced through the stricture and left in place: threads attached to the tube are brought out at the mouth, so that the tube can be removed when desired. In the later stages, when there may be a fistulous communication between the œsophagus and the air-passages, it is important, in order to avoid any lung complications, that no food at all should pass into the œsophagus; and this is effected by the patient constantly wearing a tube which is long enough to pass through the stricture into the stomach and to convey liquid food. The upper end of the tube, which is free at the back of the mouth, is fixed by threads to the whiskers, to the skin behind the ears, or to the teeth. The condition of the threads should be examined daily, and care taken that they are not bitten through; otherwise the tube may be partially swallowed, in which case it is extremely difficult, if not impossible, to recover it.

Mr. Berry has advocated the employment of soft india-rubber tubes, which may be introduced and removed on catgut guides, after the manner of railway catheters in the urethra.

Gastrostomy is usually resorted to much too late in the course of the disease, and thus the operation has not a fair chance. This is well borne out by Newman's statistics of gastrostomy both for cicatricial and malignant stricture. Of 48 cases of cicatricial stricture, 23 died within a month of the operation; and of 280 cases of gastrostomy for malignant disease, 154 died within a month. The causes of death in both classes taken together of those dying within the month were—exhaustion in 53 per cent, peritonitis in 21 per cent, pneumonia in 15 per cent.

On the whole, gastrostomy is the most satisfactory method of treatment, and should not be delayed until the patient is emaciated and pulled down by chronic starvation. Yet at best gastrostomy is but palliative, inasmuch as the growth gradually invades the important structures in immediate relation with it, and thus leads to death in one or other of the various ways already mentioned. It is therefore hardly fair to compare the effects of gastrostomy with those of colotomy for carcinoma, and to expect that life will be as much prolonged in the former case (6).

Prognosis.—Unless radically removed, and this is practically out of the question, the growth will rapidly cause death; most patients die well within a year from the beginning of symptoms. But the course of

the disease varies under different conditions, and life may be prolonged by careful feeding through a Symonds tube, or by an early gastrostomy.

When carcinoma attacks the upper part of the œsophagus its course is more rapid than when the lower part is affected.

When there is evidence of the extension of ulceration into any of the neighbouring parts, death is at hand.

Innocent Tumours.—Simple or benign tumours of the œsophagus are rare if the small warty growths, which are quite unimportant, be excepted.

Fibromas and myomas may be of considerable size, and may simulate the symptoms of cicatricial or malignant stricture; on the other hand, from compensatory dilatation of the œsophagus around the tumour (which is almost always polypoid) there may be no obstruction; and symptoms may be absent.

Other varieties of growth have been described, but are mere pathological curiosities.

Periœsophageal abscess.—As the result of caries of the spine an abscess may be formed in connection with the œsophagus after the manner of a post-pharyngeal abscess; but a periœsophageal abscess frequently presents laterally, and displaces the œsophagus to one side and not anteriorly.

Suppurating glands, whether secondary to inflammation elsewhere, as in the mouth, throat, or head, or as the result of tubercle, may lead to an abscess in close relation to the œsophagus. A caseous gland at the bifurcation of the trachea may soften down and discharge into the œsophagus, or into the trachea; or may lead to a fistulous communication between the two. Cicatrisation of the abscess cavity left after discharge into the œsophagus may result in a traction diverticulum. Suppuration and abscesses, from whatever cause they arise, may open into the œsophagus; thus an abscess in connection with the larynx or thyroid gland may discharge into it. Suppuration in the immediate neighbourhood of the œsophagus may follow acute traumatic œsophagitis, or may result from ulceration of the œsophagus.

The abscess may be of very various dimensions; eventually it discharges into the œsophagus: while eroding the œsophageal wall it may travel in the loose submucous layer, and dissect the mucous coat from the muscular coat, and thus give rise to a diffuse phlegmonous œsophagitis. Except when due to caries of bone the course of a periœsophageal abscess is acute.

Symptoms.—The chief symptoms are pain in swallowing with difficulty in taking food, and pain on movement of the neck. Dyspnœa, due to pressure on the trachea, may come on.

Treatment.—If the abscess is in the neck it should be opened at once. A periœsophageal abscess in the thorax in connection with the bronchial glands cannot be diagnosed with any certainty; but it has been suggested that emetics should be given in the hope of rupturing the abscess by the effort of vomiting.

Spasmodic stricture of the œsophagus. — **Œsophagismus** occurs in neurotic young women (18-30 years); but also in men of a similar type, and in hypochondriacs. Cases have been recorded in which it has attacked mother and daughter in such a way as to appear hereditary.

It may be purely psychical; but it is more often, perhaps, associated with reflex irritation, especially in the area supplied by the vagi, if not secondary to it. Thus, as might be expected, spasm may accompany any disease of the œsophagus itself; especially inflammation and ulceration. But in these cases, inasmuch as there is some organic change in the gullet, the condition of spasm is secondary or subordinate, and is not to be described simply as spasmodic stricture. Characteristic examples of this neurosis may seem to depend on pharyngitis or gastric disorder, and are sometimes dated back by the patient to damage received in swallowing rough food, bones, or foreign bodies; it is then perhaps due to "auto-suggestion." Reflex irritation from distant parts, such as the genital organs, appears to play some part in setting up spasm. Spasm is a prominent and painful feature in human rabies, and is present in the hysterical imitation of that disease. It has been attributed to the gouty and rheumatic diathesis.

Its *pathology* consists in an irregular contraction of the circular coat which has been supposed to precede the bolus of food instead of accompanying or following it, as normally it should do.

Spasm of the œsophagus has been associated with the abnormal origin of the right subclavian artery from the thoracic aorta. This vessel, which is due to persistence of the right 4th aortic arch, passes behind, sometimes in front of the œsophagus, and has been thought to compress that tube and thus to give rise to "dysphagia lusoria." Whether this be so appears doubtful.

Symptoms.—The dysphagia, which is more or less paroxysmal and varying in degree, comes on suddenly, and may be attended by gulping noises. There is no inclination for food, and when taken it may be suddenly and violently rejected. Spasm is increased by cold liquid food, so that the patients usually prefer warmed food. Globus hystericus may be present, and be accompanied by some pain and discomfort. As emaciation is not present, or advances to no great degree, its absence helps us to distinguish this affection from malignant stricture.

There is no constancy in the spot where the bougie is arrested; the resistance will nearly always disappear as the pressure is continued, if necessary under the influence of chloroform.

Diagnosis.—The age, sex, and character of the patient should be taken into account. The manner in which the dysphagia comes on, and its intermittent or progressive course, are important in distinguishing spasmodic from organic spasm. The absence of emaciation is strongly in favour of spasm. In addition to the knowledge to be obtained from the passage of a bougie, to which I have already referred, auscultation of the œsophagus may be employed. In spasmodic stricture the œsophageal

sound is unaltered; in organic stenosis it is prolonged, delayed, or replaced by a continuous confused bubbling sound, sometimes with a grating note.

Treatment is local and constitutional.

The passage of a bougie may bring about a cure, or cautious galvanism by means of an œsophageal bougie may be successfully employed.

Liquids, not solid food, should be given; and care should be taken to see that they are warmed and sweetened. General treatment of the patient's health and special anti-hysterical and anti-spasmodic remedies should be prescribed, and attention directed to the cure of hysteria or hypochondriasis.

Simple ulceration.—Simple ulceration of the œsophagus is rare. It may be due to damage done by rough masses of food, or by foreign bodies. In poisoning by tartar emetic multiple superficial ulcers may result. Ulceration has been met with in the course of typhoid fever. Dr. Wilks refers to two cases in which ulcers, closely resembling gastric ulcers, were found. Such ulcers near the lower end of the œsophagus have been found associated with gastric ulcers, and have been attributed to the digestive action of the regurgitated contents of the stomach occurring during life in dyspeptic persons. Ulceration is said to result from chronic œsophagitis. It is sometimes associated with syphilis; very rarely with tubercle. Ulceration in traumatic œsophagitis has been already mentioned. In newly-born infants ulceration may follow idiopathic œsophagitis, and occur either at the upper or lower end. Speaking generally, simple ulcers are more frequent at the lower end of the œsophagus, and are occasionally found post-mortem without any previous evidence of their presence. In some of these cases the ulceration may have been due to digestion occurring during the death agony or after death.

Ulcers have been described as occurring on the mucous membrane of the œsophagus in cases of pemphigus and in variola. With regard to the latter, however, considerable difference of opinion exists; and from those best qualified to decide it appears that if small-pox pustules do occur in the œsophagus the occurrence is an extremely rare event.

The *symptoms* due to ulceration are pain and difficulty in swallowing, and spasm. But the existence of ulceration can be rarely diagnosed during life, and the presence of ulcers at the autopsy is usually quite unexpected. Ulceration, by opening some of the œsophageal veins, may give rise to severe and considerable hæmorrhage.

Treatment.—The existence of simple ulcer can only be guessed at; but if it is suspected the treatment should consist in giving soft, pulpy, or liquid food by the mouth; or, if there be much dysphagia, nutrient enemas. The introduction of bougies should, of course, be avoided.

Results of simple ulceration.—Healing of a simple ulcer may give rise to cicatricial stricture of the œsophagus.

The presence of simple ulceration disposes to rupture of the œsophagus,

and has probably often preceded it. Perforation into the pleura may set up empyema; or a fistulous communication with the bronchi may result. Probably many of the cases of tracheo-œsophageal or broncho-œsophageal fistula, not of malignant origin, are due to softening of tuberculous bronchial glands putting the two tubes into communication; others are due to a perforating ulcer in the œsophagus.

Rupture.—Rupture is a very rare accident; so much so, that by some it has been regarded as only post-mortem digestion of the walls of the œsophagus from regurgitation of the contents of the stomach. But there is no doubt that the œsophagus has been ruptured as the result of violent vomiting and retching.

Etiology. — It is probable, however, that there was some local alteration in the wall of the œsophagus; either that it was weakened by ulceration at the spot where the rupture eventually occurred, or that there was some narrowing or obstruction, as, for instance, by a foreign body, above the point of rupture; so that when the contents of a full stomach were driven up into the œsophagus very forcible distension took place. The rupture always occurs in the lower part of the œsophagus, which Mackenzie has shown to be the weakest part, and is in the long axis of the tube.

Symptoms.—Agonising pain comes on directly after the rupture has presumably occurred, and is increased on movement. Though the patient is able to swallow yet, as the food passes into the pleura, he can no longer vomit it from the stomach; but he may bring up a little blood. Marked collapse supervenes, and death follows after an interval varying from a few hours to a few days. So far as we know, it is invariably fatal.

Subcutaneous emphysema has been noted in some cases as a result of rupture of the œsophagus.

The *diagnosis* is very difficult. If rupture be suspected, food should be given either by rectum or by means of a Symonds tube kept permanently in the stomach.

Varicose veins.—In portal obstruction, as is well seen in cases of cirrhosis, all communications between the radicals of the portal vein and the general systemic venous system become dilated. There is an anastomosis between the gastric and œsophageal veins, whereby the blood can pass from the stomach into the azygos veins without having to traverse the liver. Varicosity of the veins at the lower end of the œsophagus may, however, occur without there being any morbid condition of the liver. It is of course rare, but cases have been recorded in which fatal hæmorrhage in quite young patients was due to this cause. These veins may become considerably dilated and varicose. Rupture or ulceration of these œsophageal piles have led to fatal hæmatemesis. This condition, however, can scarcely be diagnosed, and at best can only be guessed at.

Treatment would be that of the primary condition, with very careful feeding and avoidance of any rough or irritating food.

NEUROSES OF THE ŒSOPHAGUS.—**Paralysis** of the œsophagus is a decidedly rare affection.

Etiology.—It may be functional or hysterical in origin; but in hysteria spasm is much commoner than paralysis. It may occur in cerebral tumour and general paralysis of the insane.

Diseases attacking the nuclei in the pons and medulla, as in bulbar paralysis, and lesions of the vagi, and as peripheral neuritis due to lead or the poison of diphtheria, may occasionally produce it. It has been thought that, like dilatation of the œsophagus, it may be due to muscular weakness, and that difficulty of swallowing occurring in the course of acute fevers may thus be explained.

Morbid anatomy.—Little is known of the morbid anatomy of the affection: Morell Mackenzie thought it probable that the lumen of the tube becomes diminished and the walls degenerated. On the analogy of other paralysed viscera dilatation would have been expected. Possibly some cases of simple dilatation of the œsophagus, where there is no apparent cause, such as a stricture, may be thus explained.

The symptoms consist in a difficulty in swallowing food, which, however, though constant, is not absolute unless the pharynx be involved. The normal œsophageal sound, as heard by auscultation, is altered or lost. Passing a bougie proves the absence of any obstruction or stricture. Regurgitation of food, which is common and copious in dilatation, seldom occurs in paralysis.

The *treatment* should, when possible, be applied to the primary cause; but local stimulation by pungent food, the passage of a tube, or electricity, may be tried. Faradisation of the interior of the œsophagus must be carried out with caution, as the vagi lie in close proximity. It is obviously more likely to succeed in the hysterical cases than in those the result of grave organic diseases of the nervous system.

In cerebral tumour or bulbar paralysis care must be taken to see that a sufficiency of food is taken, and, if need be, the patient should be fed by the stomach-tube.

H. D. ROLLESTON.

REFERENCES

1. ALLBUTT, T. CLIFFORD. *British Medical Journal*, 1875, vol. ii. p. 420.—2. BERRY, J. *St. Bartholomew's Hospital Reports*, vol. xx. p. 45.—3. BUTLIN. *Sarcoma and Carcinoma.* London.—4. *Idem. Clinical Journal*, vol. v. p. 137.—5. *Idem. Medico-Chirurgical Soc. Trans.* vol. lxxvi. p. 269.—6. BRUCE CLARKE. Treves' *System of Surgery*, vol. ii.—7. DEBOVE and ACHARD. *Manual de médecine*, tome v. p. 220.—8. DUPLAY and RECLUS. *Traité de Chirurgerie*, vol. v. p. 445.—9. EWART and BENNETT. *Clinical Society's Trans.* vol. xxiv. p. 249.—10. FOX, WILSON. *Treatise on Diseases of Lungs*, p. 1145.—11. FRANKS, W. K. *British Medical Journal*, 1894, vol. ii.—12. HARRIS, V. D. *St. Bartholomew's Hospital Reports*, 1892, p. 89.—13. MACKENZIE, MORELL. *Diseases of the Throat and Nose*, London, vol. ii. 1884.—14. NEWMAN, D. *Malignant Disease of Nose and Throat.*—15. PITT, G. N. *Trans. Path. Soc.* vol. xlvii. p. 45.—16. RICHARDSON. *Intrathoracic Auscultation. Med. Soc. Trans.* vol. xvi.—17. SHATTOCK. *Pathol. Soc. Trans.* vol. xli. p. 87.—

18. SYMONDS, C. J. *Lancet*, 1889, vol. i. p. 622.—19. TARGETT. *Path. Soc. Trans.* vol. xl. p. 76.—20. *Idem. Path. Soc. Trans.* vol. xliv. p. 48.—21. TAYLOR, F. *Guy's Hospital Reports*, 1892, p. 128.—22. THIN. *Medico-Chirurgical Soc. Trans.* vol. lxxv. p. 286.—23. VOELCKER. *Path. Soc. Trans.* vol. xlii. p. 87.—24. WETHERED. *Path. Soc. Trans.* vol. xlii. p. 116.—25. WILKS. *Pathological Anatomy*, p. 382. London, 1889.—26. WILLIAMS, R. *Diseases of the Breast*, p. 234.

H. D. R.

DISEASES OF THE STOMACH

DYSPEPSIA

DYSPEPSIA is a general name which is generally used as synonymous with indigestion. It properly means discomfort or pain during digestion in the stomach; but it is frequently used to signify discomfort or pain during the process of digestion, whether in the stomach or intestines. The vagueness of its significance has both advantages and disadvantages. The disadvantages of the name are that it signifies so many different conditions that it is little or no guide to the exact functional or organic lesions of the several organs concerned in the digestive process; and without a more or less accurate knowledge of the nature of a disease we cannot hope for success in treatment. Its advantages, on the other hand, are that it includes a group of symptoms which cannot always be referred to an exact physiological or anatomical basis. In this respect it corresponds to albuminuria or jaundice, affections which, although they can often be assigned correctly to their causes, yet sometimes defy exact diagnosis.

It may also be compared with the symptom of itching in the skin, which may be due to eczema, urticaria, pruritus; or, as in jaundice or Bright's disease, to the presence of abnormal constituents in the blood. If we had to depend for our diagnosis solely on the patient's account of his feelings, we could not distinguish these affections; nor could we do so successfully even if we were able to palpate the surface. By ocular inspection we may frequently distinguish them; but if, instead of being exposed to view, the skin were inside the body, it is evident that the diagnosis would often be impossible. Thus it is with many cases of dyspepsia. The patient complains of great discomfort, but we are unable to discover any organic cause; and frequently are not able to decide even upon the nature of the functional derangement on which the symptoms depend. In some cases the derangement is probably very slight, although the symptoms may be severe; just as itching in eczema, although the irritation is confined to the surface of the skin, may render the patient's life almost intolerable. Some cases of dyspepsia very probably depend upon a similar kind of irritation of the gastric or intestinal mucous membrane; for occasionally eczema will disappear and indigestion take its place, and conversely.

As medical knowledge advances the use of the name dyspepsia will be

greatly restricted (1), and we shall be able to assign the different forms of indigestion more and more exactly to their proper causes. At present, however, it must be retained.

Foods.—In order to understand the symptoms of dyspepsia we must first consider the various classes of foods, and the mode in which they are normally digested. In place of simply tabulating the various classes of foods, it is easier to suppose an ordinary mixed meal, consisting of beefsteak with fat attached, bread or potato, salt, pepper, mustard, pickles, fruit stewed with sugar, and alcohol in beer, wine, or spirits. The lean part of the beefsteak is a good example of the proteid constituents of food; the fat of the fatty constituents; the bread or potato of the carbohydrates; the salt of the mineral constituents, of which others are contained in the meat itself and in bread. Water is the largest and one of the most essential ingredients of the body. The stewed fruit and vegetables contain cellulose and neutral salts of vegetable acids combined with potash or lime. The sugar, like the bread and potato, belongs to the class of carbohydrates; the pickles contain organic acids with cellulose; and mustard and pepper contain pungent oils, which have the power of irritating, with more or less energy, any mucous surface with which they are brought in contact. Alcohol has a similar power of local irritation, which, however, is modified very greatly by its dilution; moreover, after its absorption, it has a marked influence on the nervous system and circulation.

Normal digestion.—Although this subject is fully dealt with in the introductory chapter (p. 281), for present purposes I may briefly sum up the main argument as follows:—In the mouth foods of all kinds ought to be thoroughly broken up by the teeth and softened by the saliva, so as to reduce the food mechanically before it is actually swallowed. Meanwhile the saliva converts starch into maltose; so that if a piece of dry bread be chewed long enough it becomes distinctly sweet in the mouth. The saliva has an alkaline reaction, and the food which has been chewed and well mixed with it in the mouth is consequently alkaline. On reaching the stomach, this alkaline material stimulates the secretion of acid gastric juice from the glands which line the walls of the stomach; and by admixture with this juice the food becomes still more fluid. The lean meat, or other proteid, unites with pepsine and hydrochloric acid, and is generally split up into albumoses and peptones. The fat undergoes hardly any chemical change; the connective tissues enveloping the fatty particles of the meat are dissolved and the fat is set free; but the dissolved albumins and albumoses in the stomach tend to emulsify it again. The starches at first undergo still further change into sugar by the continued action of the saliva; but this change is arrested after a while by the increasing acidity of the gastric juice. Cane sugar is partly left unchanged, partly converted into glucose and lævulose. The salts are partly absorbed and partly remain in the stomach; and the same is the case with the water which may be consumed with the meal. The cellulose is digested in the very slightest degree, if at all; but it may be mechanically

comminuted by the movements of the stomach, for the whole mass that has been swallowed is churned up with the gastric juice for a varying period of time. Usually part of it is discharged at the end of about two hours or two hours and a half, and the whole is usually cleared out in about five hours. The acid chyme pouring into the duodenum there stimulates the secretion of the alkaline bile and pancreatic juice, as well as of the intestinal juice; and here the starches are converted into maltose and dextrose. The fats are further emulsified and split up into glycerine and fatty acids, and partially saponified by combination with potash or soda. The albumoses undergo further change into peptones; and any cane sugar left unchanged in the stomach is converted into dextrose and lævulose. Some absorption here takes place, but this is chiefly effected in the large intestine where the movement of the bowel is slow; food, which may take only five to eight hours to pass from the stomach to the ileo-cæcal valve, usually takes sixteen hours to pass from this point to the anus.

Symptoms.—In health we are unconscious of the existence of any part of our body save in so far as we get pleasure from its exercise; and this is true of the digestive organs as well as of others. For in perfect health the only sensations connected with digestion are those relating to the ingestion of food and the ejection of excreta; and the slight inconvenience they occasion is more than compensated by sense of relief in the latter case, and the positive pleasure associated with the former.

In some cases of dyspepsia the symptoms are associated more with the palate than with the stomach; in other cases rather with the intestines. The pleasure afforded by eating is frequently referred to the palate; although it probably depends to a great extent upon the state of the stomach. In dyspepsia the normal desire for food occasionally gives place to a ravenous craving; but more frequently it leads to a loss of appetite, distaste for food, nausea, or actual vomiting. The presence of food in the stomach, instead of being unfelt as in health, gives rise to a sense of oppression in the epigastrium, disagreeable eructations of gas, food, or acid, and feelings of weight, oppression, uneasiness, discomfort, or pain. The pain may be diffused; it is very often situated at the junction of the œsophagus with the stomach opposite the end of the sternum, when it is known as heartburn. In the intestine, likewise, we may have uneasiness, pain, distension, passage of flatus, and either diarrhœa or constipation. In addition to these symptoms we sometimes have a great flow of saliva from the mouth, or the ejection of a large quantity of watery fluid, really saliva although it appears to come from the stomach. This is frequently accompanied by very acid eructations, the acid being such as to give rise to a burning in the mouth; and from the burning in the epigastrium and mouth, with which the flow of watery fluid is frequently associated, the symptom has received the name of pyrosis, a name which is now given to this ejection of water even although no feeling of burning be present.

Briefly, the symptoms of dyspepsia are a furred tongue, a bad taste

in the mouth, want of appetite or even loathing of food, nausea, vomiting, oppression in the chest, weight at the epigastrium, pain, flatulence, acidity, eructations, pyrosis, constipation, or diarrhœa. The flatulence either passes upwards in belching or downwards per anum, or, if it remain in the stomach and intestines, it gives rise to distension.

Analysis of symptoms.—*Furred Tongue.*—The tongue is usually taken as a useful index to the condition of the mucous membrane of the alimentary canal. In health it ought to be clean; but in disease it is frequently covered with a more or less thick coating of a white, yellow, brown or even black colour. This coating consists chiefly of epithelium with masses of micro-organisms. Its thickness and colour depend to a considerable extent upon the diet, and if the diet be very soft—especially if it be liquid, as in the case of the milk diet usually employed in typhoid fever—the tongue has a thick, white, creamy coating. When the diet is solid the food tends to rub off the accumulations of epithelium, and to keep the tongue clean. But the growth of microbes on the tongue is greatly stimulated by decomposing food; and this may be well seen in the case of persons with a decayed molar tooth, in which particles of food accumulate; the decomposing food in the hollow tooth seems to act as a kind of manure for the microbes on the tongue, and these consequently grow luxuriantly, giving rise to a thick patch of fur on the tongue close to the decayed tooth; even though the rest of the tongue may be clean. How far the thick fur which occurs in some cases of dyspepsia, or in fevers, is due to alteration in the secretions of the mouth, and how far to trophic changes in the mucous membrane of the tongue itself, is not certainly known. Usually, however, a white pasty fur on the tongue is looked upon as a sign of atony and weakness in the digestive tract; while a pointed tongue with large papillæ, clean and rather red at the edges and tip, is regarded as a sign of irritation and an indication for soothing treatment; a pale, flabby tongue is supposed to indicate the necessity for stimulating and tonic treatment. A bad taste in the mouth is sometimes due to decaying teeth or to artificial teeth or plates that are worn too constantly, and are not sufficiently purified by antiseptics; but in addition to this, we know that a bad taste may arise from bitter substances given as medicine. Not only does quinine produce a bitter taste in the mouth when swallowed, but if administered with iodide of potassium the bitter taste may be persistent during the whole day. The iodides alone give rise to a saline, disagreeable taste, of which many patients complain; this is caused by absorption from the stomach and re-excretion into the mouth through the salivary glands. When quinine is given at the same time the iodide appears to carry the quinine along with it into the mouth, and thus to give rise to the bitter taste. Bitter substances are formed in the body naturally, even when no medicines are taken; and although fresh healthy bile is tasteless, bile is sometimes excessively bitter. The cause of the bitterness has not been exactly ascertained; but it is probably due to accumulation of those bitter substances formed during digestion, which frequently make milk disagreeable to the palate when

artificially peptonised for too long a time. It is probable that these bitter substances reach the mouth in the same way as iodide of potassium or quinine.

Disagreeable breath.—A disagreeable smell of the breath, like a furred tongue or unpleasant taste, may be due to decomposition of food in carious teeth; but in some cases it is almost certainly due to the elimination of substances which have been absorbed from the intestine and are excreted through the lungs. Sulphuretted hydrogen, when injected into the intestine, is absorbed with great rapidity and eliminated through the lungs; and the same is the case with such vegetable volatile oils as those of onion and garlic.

Appetite, although closely associated with the gratification of the gustatory nerves in the mouth, is yet more intimately dependent upon the condition of the stomach, and upon the wants of the body as a whole. The exact cause of it has not yet been ascertained, but certainly it is closely connected with the circulation of the blood in the stomach. The mucous membrane of this organ during fasting was observed by Beaumont to be pale; but on the ingestion of food it became rosy at once, and secreted gastric juice. It is probable that appetite consists in a condition of the nerves of the stomach similar to that which is produced in the nerves of the skin by itching or gentle tickling; and is not improbably connected, to some extent, with distension of the lymph spaces in the mucous membrane, just as in the skin a sensation of itching frequently precedes the outbreak of sweat. When the stomach is somewhat weak or atonic there is frequently no desire for food at first; but after a little has been taken the appetite comes with the eating until a fair quantity has been consumed. Sometimes Beaumont observed that a craving appetite was associated with a red and irritable mucous membrane on which even some abrasion of the surface was present. When, however, irritation of the stomach, mechanical or chemical, is carried too far, the normal secretion of gastric juice stops, the circulation becomes lessened, the mucous membrane turns white, and mucus is secreted. Still further irritation causes retching and vomiting.

It is evident that, if appetite be thus closely associated with the condition of the circulation in the stomach itself, the appetite and the circulation together are likely to be influenced by the state of the liver; for nearly all the blood from the stomach must pass through the liver before it can return into the general circulation. If this organ be congested, and the circulation through it slow, the appetite is likely to fall into abeyance. It must be borne in mind also that various substances have the power of stimulating the walls of the stomach in such a way as to produce increased appetite, anorexia, or vomiting. Many substances which in small doses increase the appetite are emetics when given in large doses. Thus a little mustard or horse-radish, a little salt or other condiment taken with food may render it more palatable; a bitter tonic taken before a meal will increase the appetite; but all these substances taken in large quantities act as emetics. A very minute quantity

of arsenic is a powerful stimulant to the appetite, but a large quantity produces most violent vomiting.

In dyspepsia, then, we may have a craving appetite, a want of appetite, or an appetite for abnormal kinds of food. The craving appetite, according to Beaumont's experiments, appears to go with an irritable condition of the stomach, such as might be caused by small doses of arsenic. It is very frequently noticeable as the first stage of what is called "a bilious attack"; and when the irritation in the stomach becomes greater, the craving appetite gives place to anorexia, and is followed by nausea, or even by vomiting. Lack of appetite may be associated with two conditions: either with the absence of tone and deficient circulation through the stomach, or with excessive irritation bordering upon nausea. In the first case, where lack of appetite is dependent upon weakness and absence of circulation, the person has little or no desire to take food; but after beginning to eat, the gastric circulation becomes brisker, appetite comes with eating, and the person may be able to take a fair meal. In the case of a craving appetite, however, things are different; and the stimulation by food, which in the case just mentioned brings on the appetite, stimulates the irritable stomach to excess, so that appetite very quickly disappears; if, then, more food be forced down, nausea or vomiting ensues. Where the stomach is already so irritable that nausea is felt before the food is taken, vomiting is very likely to come on immediately after eating. A lack of appetite restored by eating, therefore, indicates an atony of the stomach, which is likely to be benefited by tonics and stimulants; a craving appetite easily satisfied, or anorexia with a tendency to nausea in eating, indicates irritation and requires sedative treatment.

It must be remembered that various poisons injected into the veins are excreted by the walls of the stomach; thus tartar emetic, morphine, and serpent's venom injected into the veins are all excreted by the gastric mucous membrane; and the same is the case with poisons formed in the body, such as the toxalbumins of cholera, and possibly also toxalbumins in cases of renal disease.

It is of the utmost importance to bear in mind that disturbance of the appetite may be due to poisons formed in the intestine or in the tissues and excreted into the stomach, where they give rise to disturbance; as we observed that they do in the mouth. The elimination of these poisons from the body is, therefore, to be carefully attended to as a means of restoring appetite.

Vomiting is the expulsion of the contents of the stomach through the œsophagus and mouth. It is effected by the squeezing of the stomach between the diaphragm and abdominal walls. This squeezing is ineffectual to empty the stomach if the cardiac sphincter be contracted; when the movements are simply known under the title of "retching." When the cardiac orifice dilates, and simultaneously the diaphragm descends to its utmost, and the abdominal muscles contract, the contents of the stomach find an issue through the œsophagus, and vomiting occurs.

The nerve-centre by which the movements of the stomach, diaphragm, and abdominal muscles are so co-ordinated as to produce vomiting is situated in the medulla oblongata, and is closely associated with the respiratory centre; some nerve-cells being in all probability common to both centres. The vomiting centre may be stimulated sometimes by poisons, such as tartar emetic or apomorphine which act upon it directly; but it is usually brought into action reflexly by irritation which may start from various parts of the body, and is conveyed to it by the afferent nerves. When stimulated from the brain, as in violent emotion or in organic disease—such as tubercular meningitis or cerebral tumour—the vomiting is sudden and not associated with nausea; and the same is the case when it is stimulated from the pharynx by the tickling of a feather. Even stimulation from the stomach may be associated with very little nausea; mustard and hot water cause vomiting speedily, but with very little antecedent nausea; tartar emetic, on the other hand, causes much nausea. Vomiting is associated also with irritation in the liver, kidney, intestines, and genital organs; so that during the passage of a gall-stone or of a renal calculus there may be violent vomiting; and when vomiting is persistent the presence of a hernia should always be looked for. The vomiting of pregnancy is usually supposed to be reflex from the uterus, but it may be due to the presence of toxalbumins, as in cholera. Vomiting in dyspepsia is usually due either to the condition of the contents of the stomach or to that of the gastric walls. Thus it may occur after too bulky a meal, when the food, although perfectly wholesome, has been taken in such large quantity that the stomach is unable to digest it. Or it may occur from the ingestion of food which is either irritating in itself, or is apt to undergo such decomposition as to lead to the formation of irritating products.

Thus, tainted meat or fish, an oyster of doubtful quality, or bad mushrooms may cause vomiting very shortly after their introduction; while a mixture of pâté de foie gras with milk and beer may yield irritant products, and give rise to vomiting some hours later; although each article was good in itself, and the quantity taken not excessive. The food which has been taken into the stomach is much more liable to undergo decomposition with formation of irritating products if delayed in the stomach. This is especially noticeable in cases of contracted pylorus, whether in consequence of malignant disease or from cicatricial contraction due to old ulcers. In such cases the food remains many hours in the dilated stomach, and is then vomited; usually in a state of fermentation. But even in health imperfectly masticated food may remain too long in the stomach, as the lumps in it are not readily dissolved by the gastric juice. When these lumps press towards the pylorus they stimulate it to contraction, when not only is their own passage arrested, but a quantity of other food also is kept back with them. Thus, pieces of potato, of apple, of cheese, or even of butcher's meat, which have been swallowed without being comminuted by the teeth, are again and again forced to the pylorus by contractions of the stomach; but not being able to get through, they

are ultimately rejected by vomiting. In cases of sick headache, a transient condition of dilatation of the stomach, and probably of contraction of the pylorus, occurs—the condition being, in fact, one of a proximal dilatation with peripheral contraction, similar to that which I have pointed out in the arteries of the head during migraine.

The condition of the gastric walls is a most important factor in vomiting. When the mucous membrane of the stomach is rubbed too hard with a piece of glass rod, the natural rose colour disappears, the secretion of gastric juice stops, mucus is secreted, and vomiting is set up. Here the vomiting is, no doubt, simply reflex, as in the case of vomiting by tickling the fauces with a feather; but in Beaumont's observations on Alexis St. Martin we notice that great irritability of the mucous membrane of the stomach tended to give rise to nausea and vomiting on taking food.

In some cases, no doubt, there is hyperæsthesia of the mucous membrane of the stomach, just as there may be of the fauces. The condition of the stomach in sick headache has already been noticed. A common cause of vomiting in the morning is the practice of taking an excessive quantity of spirits at night before going to bed. In life assurance it is of great importance to ascertain the habits of the proposer in regard to alcoholic liquors; and as answers to direct questions on this point are apt to be sometimes evasive, if not untrue, some medical examiners are in the habit of asking whether the patient feels sick in the morning just after rising, or if he has got a good appetite for breakfast. Inability to eat breakfast, and morning vomiting especially, are regarded as suspicious signs, and as indications that the life is a doubtful one for insurance purposes.

Oppression.—The feeling of oppression, which is generally referred to the cardiac region, is in most cases due to distension of the stomach with flatulence; but in some is due no doubt to reflex irritation of the vagus acting upon the heart: indeed, the pulse-rate may actually be much slowed by gastric irritation.

Pain in the stomach varies very much both in extent and character. In simple dyspepsia, not associated with ulceration or malignant disease, it is usually either of a dull character, extending over the whole epigastrium, or is a burning sensation nearly opposite the end of the sternum. The burning is closely associated with great acidity of the contents of the stomach, so that, when these regurgitate into the mouth, they seem to give rise to a burning feeling in the throat and set the teeth on edge. The point at which this burning is felt is just at the junction of the œsophagus with the stomach; and I am inclined to think that it is due to irritation not of the stomach itself but of the lower end of the œsophagus by the acid contents of the stomach.

Acidity.—I have observed that when there is much acid in the stomach this pain may sometimes be brought on or relieved at will, simply by turning from one side to the other; on turning to the left side, so that the gastric contents fill the fundus and do not reach the cardiac

orifice, the pain may disappear; on turning to the right side, so that the contents of the stomach fill the smaller pyloric end, and are likely thus to reach the cardiac orifice, the burning sensation may at once reappear. I have noticed, too, that the burning sensation may come on almost immediately after drinking a cup of tea, although this liquid is neutral in itself and not irritating to the œsophagus. The tea may act simply by increasing the volume of the contents of the stomach, so as to make them reach the cardiac orifice, and irritate it by their acidity; but it seems very improbable that during the natural movements of the stomach the gastric contents should not pass over the cardiac orifice; it seems to me more probable that tea causes a protrusion of the mucous membrane of the œsophagus into the stomach, much as the mucous membrane of the rectum occasionally protrudes through the anus after the administration of an enema; the mucous membrane of the œsophagus, which is much more sensitive than the mucous membrane of the stomach itself, would thus be exposed to irritation by the acid contents of the stomach, and would feel the burning. When we swallow anything hot, the burning which it occasions ceases just at the point where the hot liquid or solid leaves the œsophagus and passes into the stomach.

This acidity of the gastric contents is not generally due to any excess of hydrochloric acid. Such an excess may occasionally be present; but, as a rule, the acidity is due to organic acids, such as lactic and sometimes butyric acids. Butyric acid seems to have an especially irritating local action. Although lactic acid appears to be a normal constituent of the gastric juice, yet excess either of it or of any other organic acid is generally due to fermentation of sugars, alcohols, or fats.

Pyroses.—Acidity in the stomach gives rise to too frequent desire to swallow saliva, which, by its alkalinity, thus eases the irritation of the lower end of the œsophagus. Not only so, but a reflex secretion of saliva appears to be induced in the salivary glands by gastric irritation; so that the saliva either runs from the mouth or is swallowed frequently, and accumulates either in the stomach or in the lower part of the œsophagus, and is then ejected in considerable quantities as a nearly colourless, slightly alkaline, and opalescent fluid.

Flatulence.—Wind in the stomach and intestines may be due either to increased ingestion, increased formation, diminished absorption, or lessened expulsion of gas. A small quantity of air is usually swallowed with the food, and a good deal is swallowed with saliva. Thus, if irritation in the stomach lead to frequent swallowing of saliva, more air is swallowed with it, and either gives rise to distension or to belching. Along with this wind small quantities of liquid or of food may come up into the mouth.

It is evident that anything that lessens fermentation in the stomach, and thus stops the formation of acid, will also lessen the flatulence which is due to the frequent deglutition of saliva. The air which is swallowed, when it is not again brought up, undergoes partial absorption, the oxygen

being quickly absorbed, while the nitrogen remains in the stomach and intestines. There is, however, another source of gas in the decomposition of food, and especially of the carbohydrates, which gives rise to the formation of marsh gas. This gas may be derived from both sugars and starches, but it is yielded in larger quantities by cellulose; so that particular articles of diet containing much cellulose are apt to cause flatulence, and when sulphur also is present—as it is in most of the cruciferæ, such as cabbage—sulphuretted hydrogen may also be found.

The occurrence of constipation and diarrhœa from changes in the intestine will be discussed later.

Causation.—The causes of dyspepsia may be divided into (A) those which concern food, and (B) those which concern the organism. The food may be wrong either in quantity or quality.

Quantity.—If food be habitually taken in too large amounts, or at too frequent intervals, the digestive organs will be unable to cope with it, and indigestion will ensue. The quantity which different persons are capable of consuming varies within wide limits; one man may eat in a day as much as would serve another for a week. Moreover, the quantity that can be consumed by the same individual varies very much under different circumstances, as with the time of the year, exposure to cold or heat, and, more especially, the amount of exercise which can be taken, and the nature of the exercise, whether it be in a confined place or in the open air.

Meal-times.—The times at which food is taken may also lead to indigestion; not only may the meals be altogether too frequent, but they may be too frequent at one time of the day and too far apart at another. Some patients, for instance, take all the food of the twenty-four hours between 8 A.M. and 4 P.M., and nothing else during the rest of the twenty-four hours; others, again, take their meals with great irregularity, sometimes lunching at 1 o'clock, sometimes fasting from 7 in the morning till 9 at night. Even if there be no such irregularity as this, the time of meals may vary from half an hour to an hour and a half.

Temperature.—The temperature at which food or drink is taken may also be productive of dyspepsia. Thus, iced water, by chilling the stomach and lessening both its secretion and movement, may cause indigestion; and very hot tea may possibly have a similar effect.

Disintegration.—The mechanical disintegration of food has much to do with its ready digestion; and when it either is not or cannot be thoroughly disintegrated it will be slow of digestion. If any one wishes to convince himself of this, let him repeat the test for pepsine in the British Pharmacopœia, using for comparison in one glass hard-boiled egg, in pieces as large as can conveniently be inserted, with pepsine and acid, and in another glass pieces of the same egg rubbed up into a fine powder. He will then easily understand how slowly large lumps are digested in comparison with the same substance when finely comminuted. One great cause of imperfect comminution is hurry at meals. The pressure of business frequently compels people to spend as little time as

possible over their food, and thus it is bolted rather than swallowed, and is masticated very imperfectly indeed. I have seen a man stick his fork into a new potato, about an inch long and half an inch across, swallow it whole at one gulp, and then complain that new potatoes are indigestible. Another cause of imperfect mastication is decay or absence of the molar teeth. On account of decayed and tender teeth on one side of the mouth the whole of the work may have to be done by the grinders on the other side. If the molars be tender on both sides, mastication must be done by the incisors, and that imperfectly; or perhaps it is not done at all. Not infrequently we find, too, that the molars, if not entirely absent, are deficient in such a way as to have almost no grinding surface. When the two surfaces meet one another over so small an area they do practically nothing more than keep the jaws apart and allow the food to escape between them.

Quality of food.—Another condition is the quality of the food. Thus flesh of various kinds may be hard and difficult to masticate; either from the age of the animal, from the meat having been cooked while still in a state of rigor mortis, or from artificial hardening by salting or smoking. If any one will take a piece of beef and tear the fibres apart, he will readily see why it is less digestible than mutton; for while beef tears into cords, mutton tears into fine threads: in the breast of a fowl the threads are still finer. In fish the fibres are not only fine, but shorter than those of flesh or fowl; and in small fish, such as whiting and smelt, the fibres are finer than in such fish as cod. Properly boiled meat or fish is softer and more easily disintegrated than the same meat or fish roasted, broiled, or fried. Although the breast of a fowl is easily broken up, the same is not the case with the leg; and the leg of a tough old fowl may be very much harder to masticate than a slice of roast beef or mutton. Skin is hard to masticate, whether it be the skin of fish, flesh, or fowl; it is hard to digest, therefore, and the same is the case with sinews and ligaments. In small fish, such as whitebait, the skin cannot be removed, nor can the bones; and the presence of the skin and the bone together makes such fish very indigestible.

Fish, flesh, or game that has become high or tainted is indigestible from a different cause; namely, from the presence in it of poisons formed by the decomposition of albuminous substances which may give rise to violent local disturbance of the digestive canal, or to symptoms of general poisoning. Curries, although very palatable, are somewhat dangerous, because the pieces of which they are composed are apt to be eaten without thorough mastication; and occasionally splinters of bone may be swallowed. Some kinds of farinaceous food are by no means easy to disintegrate; as, for example, soft rolls and buns, and newly-baked bread. Any one can see this for himself by contrasting the behaviour of a piece of new bread with the same bread when stale. If new bread be rolled between the finger and thumb it becomes a dense, coherent mass; the stale bread treated in the same way is disintegrated into fine powder. A piece of dry toast is brittle, and breaks readily into powder between the

teeth, and its brittleness is not impaired by spreading butter upon it just before eating; but if the same toast, with butter spread upon it, be laid before the fire so that the butter soaks in, it becomes much tougher and less easily broken up: thus to stand in the heat with butter upon it may convert the toast from a readily digestible into an indigestible article of food.

Pastry is usually put down as very indigestible; and the pastry of the pastry-cook is certainly very apt to cause dyspepsia; but pastry carefully made at home may often be eaten with impunity. The reason of this probably is that if the pastry is made with the very best butter, and rolled very fine so as to make it flaky, it is neither injurious mechanically nor chemically; but if it is heavy it will not be readily broken up, and if the abominations sometimes known as "cooking-butter" are used in its composition, it will be very apt to disagree chemically. Many people think that a piece of bad butter, which it would be impossible to eat if it were spread upon a piece of toast, will do no harm if incorporated in pastry, because it is not so easily detected by the sense of taste. It is true that it thus escapes the very sense which was intended to act as a sentinel to the stomach; but it is none the less injurious, and is exceedingly likely to cause sickness; butyric acid being, as already mentioned, excessively irritating to the stomach. Another frequent cause of dyspepsia is want of cleanliness in the cooking utensils. The changes which such a want of cleanliness produces in the food prepared in them are probably of so subtle a nature as at present to elude the research of the chemist; but, unfortunately, they act only too readily upon the human organism, and I believe that many a family suffers from dyspepsia simply because their cooks do not take proper pains to clean the pots and pans sufficiently soon after use to prevent decomposition of the fat used in cooking. Bad cooking is also responsible for many cases of dyspepsia in another way. When food is well cooked and appetising, the pleasure that is given to the palate excites reflexly the secretion of saliva, which does its part in digesting the starchy part of the food. Moreover, the increased amount of saliva stimulates the secretion from the stomach, and this in turn stimulates the intestines; so that the appetising character of the food is a great aid to rapid and thorough digestion. If the food, on the other hand, is badly cooked and unappetising, so far from making the mouth water and stimulating the digestive processes in the way just mentioned, it tends rather to cause disgust, and through the nervous system to stop secretion of gastric juice, and even to produce nausea.

But even when foods are properly cooked they may disagree from their chemical composition, and give rise to acidity, flatulence and other discomfort. Thus oily fish, such as herring, mackerel, eels and salmon, may disagree from their tendency to become decomposed in the stomach, and to give rise to irritating products.

It has already been said that fats undergo little or no digestion in the stomach, and that the proper place for their digestion is the intestines; but sometimes they may be split up in the stomach, probably by means

of microbes, setting free fatty acids which are exceedingly irritating; allusion has previously been made to the excessively irritating properties of butyric acid. Many children are quite unable to eat hot mutton fat without being sick, and yet the same children may be able to eat cold mutton fat without harm. The probable explanation of this is that the hot mutton fat undergoes a certain amount of decomposition in the stomach, and yields irritating products which cause sickness and vomiting; whereas the cold mutton fat, being harder, is less changed until it arrives in the intestine, where it undergoes proper digestion. Raw vegetables, as a rule, do not irritate the stomach unless they have been swallowed without thorough mastication; but they very frequently occasion great development of flatulence in the intestines. Unripe fruit not infrequently causes irritation both in the stomach and intestines, because the cellulose of which it is chiefly composed is very indigestible, and is attacked very slowly, if at all, by the digestive juices; moreover, unripe fruits are often simply crushed between the teeth and swallowed, in pieces of considerable size, without undergoing thorough mastication. Over-ripe fruit, or rather decaying fruit, is productive of discomfort from a different cause: although soft, and thus not likely to occasion mechanical irritation, it frequently contains quantities of microbes which give rise to decomposition, with the evolution of gas and production of irritating products which cause diarrhœa.

Drinks.—Beer is a common cause of dyspepsia, more especially if it be new and the fermentation not thoroughly completed. Such beer undergoes further fermentation in the stomach and intestines, gives rise to acute indigestion, and, if its use be long continued, to chronic catarrh.

Vinegar taken in large quantities leads to chronic dyspepsia with consequent emaciation, so that it is sometimes taken as a means of reducing obesity. It is a most dangerous remedy for this purpose, and I have seen its use by a well-nourished girl lead not only to emaciation, but to phthisis consequent upon malnutrition.

Acid wines (*vins aigres*) have a similar action, and, if used regularly, tend to produce gastric catarrh. Spirits in excess are also most injurious; and whisky and water (or brandy and water) at night, by its irritating action on the empty stomach, is very likely to produce sickness and vomiting in the morning.

Tea is another article of diet which is apt to cause dyspepsia; partly by its action on the stomach itself and partly on the nervous system. An infusion of tea contains a good deal of tannin, which, if taken along with butcher's meat, tends to harden the fibre and render it less digestible. Common experience has led people to avoid taking butcher's meat and tea at the same time, and rather to take with tea farinaceous food and easily-digested proteids, such as boiled eggs or fish. High teas, in which butcher's meat is taken in considerable quantity with tea as a beverage, are a frequent source of severe and persistent dyspepsia. The effect of the tea upon the digestion depends a good deal upon the quantity used,

its temperature, the frequency with which it is taken, and the kind of tea employed. A very strong infusion, especially when the water is allowed to stand on the leaves for a long time, is most injurious; and if it be taken scalding hot the effects are still worse. Constant flatulence, pain in the stomach, and emaciation are frequently noticed in poor women who come as hospital patients. Many of them keep the teapot on the stove the whole day long, so that the tea is constantly stewing, and what they drink is rather a decoction than an infusion. It is best to allow the tea not to stand for more than three minutes upon the leaves and then to pour it off; and in some cases the plan recommended by the late Sir Andrew Clark of infusing the tea with milk instead of water seems to be very useful. If the water be very hard, effervescing water, such as soda or potash water, may be employed instead of well water. Some patients bear China tea better than the teas of India or Ceylon. Tea-tasters tell me that although all the tea plants come originally from China, yet the tea plants have been grown in the same soil for centuries in China; for about fifty or sixty years in Assam, and for about twenty years in Ceylon. But the Ceylon and Indian teas are really drunk much stronger than China teas; many people judge of the strength of an infusion simply by its colour, and as the China teas yield more colouring matter than Ceylon or Assam teas, they contain with a good depth of colour less of the other ingredients than Assam and Ceylon teas. These latter teas should be drunk in a pale straw-coloured infusion instead of the dark brown infusion of the China tea, as the pale infusion of the former corresponds in strength to a dark infusion of the latter.

The quantity of fluid taken at meals is often of importance. In the case of tea a chemical action of the liquid interferes with digestion; but even too much water with meals may cause dyspepsia. One reason of this is that if water be frequently sipped during the meal the food is apt to be softened in the mouth by water instead of by saliva, and thus passes into the stomach imperfectly masticated, and with its farinaceous ingredients little changed by the saliva. The fluid also dilutes the gastric juice and renders digestion in the stomach slow, so that time is afforded for the development of microbes, the growth of which is further favoured by the dilution of the gastric juice which in its normal concentration has an antiseptic action. Dyspeptics are frequently advised not to drink during meals, but to drink freely after they have finished their meal. This advice is good so far as it goes, because it prevents the patient from washing his food down without either mastication or insalivation; but it does not prevent dilution of the gastric juice. A better plan is to drink water, and especially hot water, an hour before meals.

Microbes.—Imperfectly cleaned dental plates or carious teeth may form a breeding-ground for microbes which are carried down from the mouth into the stomach. Frequent swallowing of saliva certainly seems to give rise to dyspepsia occasionally, and possibly the dyspepsia which has been observed along with dirty dental plates may really have been

due to profuse salivary secretion caused by the irritation they produce. Such secretion may also be induced by tobacco, either smoked or chewed, and this also leads to dyspepsia; while the action of the tobacco itself upon the nervous system, in some cases, is distinctly injurious; in others, however, if used in moderation, it may on the whole be beneficial. I have seen at least one case of dyspepsia in which everything failed to give relief until a naso-pharyngeal catarrh, from which the patient suffered, and which gave rise to constant swallowing of mucus, was treated by the removal of some adenoids; after this operation the dyspepsia disappeared.

Nervous influences.—The effect of emotion upon the stomach is well known. A piece of bad news takes away the appetite, and may even bring on sickness. Mental work immediately after a meal is very apt to disturb digestion, and if carried on regularly may lead to dyspepsia. I was once staying at a hydropathic establishment near a large commercial town when I observed that one of the rules was that any patient taking up a newspaper within an hour after dinner was fined one shilling. The reason for this was that the patients who came there were chiefly engaged in business, and the first thing they turned to in a newspaper was the money column; thus their minds became occupied with commercial affairs after meals and digestion was not so good. Robert Mayow put the case very well more than two hundred years ago. It was then supposed that all the functions of the body were carried on by vital spirits,—little imps which rushed hither and thither according to the work required in one part of the body or in another. He said that if the vital spirits are in the stomach digesting food they cannot be up in the brain, and, therefore, immediately after a heavy meal people are stupid and languid; if the vital spirits leave the stomach and go up to the brain, the digestion is not performed. If we put the word "blood" instead of vital spirits, Mayow's doctrine corresponds very nearly with that of modern physiologists. Excessive weariness tends to cause indigestion, because the wearied stomach and nervous system do not respond to the stimulus of food. Worry, anxiety, disappointments, and especially love affairs, all tend to cause dyspepsia [*vide* p. 648].

Exposure to *cold*, probably by disturbing the balance of the circulation, causes dyspepsia; and many people who are perfectly well during dry weather begin to suffer from dyspepsia as soon as the weather becomes cold and damp.

Want of exercise.—General atony of the body, such as results from lack of exercise, brings about atony of the stomach as well; and the imperfect combustion or elimination of waste products not only lessens appetite but interferes with the digestive processes. Atony of the muscles of the abdominal wall, by rendering them feeble, prevents their giving that support to the stomach and abdominal viscera which is necessary to keep them perfectly in position: the stomach and intestines, therefore, tend to fall, and we get the condition known as gastroptosis and enteroptosis [*vide* art. "Enteroptosis"]. In some cases these

conditions are increased and perpetuated by adhesions of the omentum from old inflammation, especially about the cæcum or appendix.

Adhesions of the intestines themselves also tend to interfere with the digestion, and give rise to flatulence, pain, and other discomfort. Pains which are probably due to old adhesions are sometimes very troublesome indeed [*vide* vol. ii. p. 887]. The patient wakes early in the morning with pain in the abdomen of a weary, heavy kind, and this may continue for one or two hours. It may be relieved by the passage of flatus, and sometimes disappears entirely as soon as the patient gets up. In other instances the patient suffers from it while upright and is relieved by lying down. The pains are affected by exposure to cold, by changes of weather as well as by indiscretions of diet; and they seem really to be of a gouty or rheumatic origin, and may coexist with or alternate with headache or pains in the joints. [See also art. "Enteroptosis" in this volume.]

In some cases a *floating kidney* seems to be productive of no injury whatever; in others it causes constant dyspepsia, languor, depression, inability to work, and discomfort so great as to render life a burden to the patient. [*Vide* art. on "Movable Kidney," vol. iv.]

There is one cause of dyspepsia which must be noted with great care, and this is incipient *phthisis*. There can be little doubt that chronic dyspepsia, by weakening the resistance of the organism, renders it less able to resist the attacks of microbes, and thus renders the individual more liable to phthisis; but, at the same time, the tuberculous disease in the lung itself appears to lead secondarily to dyspepsia. Whether this be due to a reflex action from the lung upon the stomach through the vagus, to toxins or toxalbumins, or to some other cause, I cannot say; but in all cases of dyspepsia, especially those occurring in patients from seventeen to twenty-five, the lungs should be carefully examined [*vide* p. 493].

Consequences of dyspepsia.—Patients suffering from dyspepsia are very apt to treat themselves, to cut off one article of diet after another, often without success, and in this way to lessen their nutrition and diminish their strength without adding to their comfort. The constant irritation which they experience from discomfort or pain is apt to lessen their power of attending to other things; so that they lose interest in outside circumstances, have less power of attention, and are tempted to concentrate their thoughts upon themselves. When they attempt to read or think they feel dull and heavy, and they do not either comprehend so readily or remember so distinctly as they did when well. Moreover, they are likely to become irritable and snappish, and such fits of irritability often alternate with feelings of depression and languor. They sometimes also complain of singing in the ears and giddiness, and of a tendency to fall. These symptoms are more frequent in elderly people, and are probably associated to a considerable extent with atheroma. When they occur in younger persons free from organic disease they are more suggestive of excessive tobacco-smoking.

Treatment.—In the treatment of dyspepsia it is important to lay

down rules for the patient when food should be eaten, how food should be eaten, and what food should be eaten. The general experience of mankind shows that four or five hours should intervene between meals; but under certain conditions it is advisable to have something to eat every two hours. In Baedeker's *Guide*, under general directions to travellers in the Alps, he advises that something should be eaten every two hours in order to avoid the fatigue that might otherwise come on. In fever, when the waste of the body is as great or even greater than in such violent exertion as Alpine climbing, every two hours is the time usually suitable for the administration of food; but when given so frequently as this it should be taken in small quantity; if much be taken at such short intervals indigestion will probably ensue.

How the food should be eaten.—It is evident, then, that food should be eaten slowly, and should be thoroughly masticated and insalivated before it is swallowed. I repeat that hurry at meals is a frequent cause of dyspepsia, and that hurry, worry, or mental exertion immediately after meals is another. In dyspeptics a certain amount of time should be allowed between the meal and the return to work, whether bodily or mental. Rest before meals is also an important factor, especially rest for a few minutes just before the evening meal, whether it be called dinner or supper; for this is the time at which the body in general and the stomach in particular are likely to be exhausted.

I have already said that much fluid at a meal, by diluting the saliva and gastric juice, is disadvantageous. It is not so injurious when taken immediately after a meal; but the best time to take liquid is an hour before meals, and the best form in which it can be taken is hot water. By drinking a tumbler of hot water an hour or so before meals, not only are the remnants of the previous meal washed out of the stomach, but also much of the water becomes absorbed. This prevents thirst being felt, obviates the necessity for drinking at meals, and, instead of the gastric juice being diluted by the water which would then be drunk, and digestion retarded, the water already absorbed supplies material for the free secretion of saliva and gastric juice, and thus accelerates digestion. If the stomach is actually dilated, and especially if this be due to pyloric obstruction, the hot water will remain in the stomach for more than an hour after it has been drunk, and instead of accelerating digestion will interfere with it. In such cases the long interval which usually elapses between dinner, supper, and breakfast may allow the stomach to become empty, and breakfast may be eaten with relish and digested with comfort. But under such conditions a tumbler of water either hot or cold, taken on rising and retained, may prevent digestion and do harm instead of good.

The experience of mankind has shown that it is advisable, to a certain extent, to separate the proteid from the farinaceous meals; a very common plan is to have a farinaceous meal with a small quantity of proteid for breakfast, a proteid meal at luncheon, a very little farinaceous food in the afternoon, and again a proteid meal in the evening. Thus,

we find that at breakfast bread and toast with some easily digested proteid —such as egg or fish—is very commonly taken; or a small quantity of a sparingly digestible proteid such as fried bacon, of which the hydrocarbonaceous fat frequently forms a large portion. At lunch the main part of the meal consists of meat, although a moderate quantity of bread or vegetables may be taken with it. The afternoon meal consists of a little bread and butter, and in the evening there follows another proteid meal like that of lunch. In cases of dyspepsia it is often useful to separate the proteids from the farinaceous foods more completely. The time of digestion of the two is different, and the parts of the digestive canal in which digestion takes place also differ.

For dyspeptics, a breakfast consisting of dry toast, rusk, or stale bread, with a little butter, which ought to be of the very best quality, should form the staple of the meal. If the digestion be very bad, the bread may have some hot milk poured upon it and be eaten in this way without butter; but ordinarily more milk, either warm or cold, may be taken with the toast, and many people without injury can take with the milk sufficient tea, coffee, or cocoa to flavour it. If the stomach be able to bear it, a soft-boiled egg or a piece of fish may be added. The mid-day meal should be chiefly proteid, such as fish, fowl, eggs or meat, with some stale bread. One of the lightest fishes is boiled whiting, for the fibre is very soft and easily disintegrated. Cod is also good, but is somewhat harder and requires more careful mastication. Boiled or broiled sole is also excellent. If the sole be fried the skin must be very carefully removed as well as the bones. Sauces, containing fluid fats, are apt to disagree; and it is safer to take the fish simply with salt and stale bread. In many cases it is better rather to spread a little good butter upon the bread and take that with the fish than to use sauce or melted butter. In the same way fowl may be eaten with stale bread. In some cases eggs in the form of an omelette are tolerated, but in many patients eggs are liable to cause biliousness. A cut from the joint, a chop, or a steak sometimes suit well, provided they be thoroughly masticated and all the stringy parts avoided. It is best, as I have already said, to drink hot water an hour before lunch; but sometimes dyspeptics will not or cannot do this, and such persons may be allowed half a tumbler of water, which should be sipped after the lunch is over. In many cases it is advantageous to add to it one or two tablespoonfuls of old whisky or brandy. Effervescent water to many persons is more palatable and more stimulating than plain water; and it may be used provided it do not give rise to flatulent distension, as in many cases it does. As a rule, wines and beers do not agree so well with dyspeptics as spirits, but it must be borne in mind that as spirits tell less upon the stomach and more upon the liver, while wine and beer tell more upon the stomach and intestine, the consumption of the former is more liable to abuse than the latter; the patient who uses spirits is longer in finding out the mischief of excessive indulgence than he who uses wine or beer. In the afternoon hot water may be drunk again, flavoured by a

piece of lemon floated upon it; and a small piece of bread or biscuit may be taken with it. Weak tea is more palatable, or milk and water, and if found not to disagree, either the weak tea, or milk flavoured with tea, may be used in the place of hot water. For the evening meal at seven or half-past seven similar food should be taken to that of the luncheon.

If the dyspepsia be less severe the patient may return to more ordinary diet, mixing the proteids and farinaceous foods in larger quantity; but care must be taken by all dyspeptics to avoid too much sugar, which is very apt to create acidity. A good general rule also is to avoid all skins and bones, and all strings, stones, or seeds. The bones of fish, and the chips of bone that occasionally occur in curries or hash, are very indigestible; so are skins of every kind, whether they be skins of fish, flesh, fowl, fruit or vegetable. All stringy meats, fruits and vegetables should be avoided, and stones and seeds are wholly indigestible.

In regard to mastication an excellent rule is that of the late Sir Andrew Clark. The mouth contains, or ought to contain, thirty-two teeth, and to every mouthful of food thirty-two bites should be given. If the teeth are imperfect, even this number is not sufficient; and as many as sixty-four or ninety-six bites may be required properly to comminute a single mouthful of steak or chop. Another rule, which is sometimes of great service in dyspepsia, is to insist that before the food is swallowed it should be so finely masticated, and so thoroughly mixed with saliva, that it shall be of the consistence of cream, and would pass through a sieve without leaving remainder. It is very much easier, however, to give such a rule than to get it followed; and it is only in very severe cases, and especially in those where the pain is so great as to suggest the presence of gastric ulcer, that patients will follow it. They often find the rule so troublesome that mere inconvenience or discomfort will not force them to follow it; and they can only be induced to do so by finding that obedience to it prevents the occurrence of severe pain, while the neglect of it causes the pain to return.

Warmth and support. Regimen.—Dyspepsia often results from undue exposure. There are four places that require special warmth, namely, the back of the neck, the front of the abdomen, the shins and the feet. Many persons, after sitting in the draught which always exists in cold weather between the door and the fireplace, will suffer from dyspepsia, and then wonder what article of food has disagreed with them. This draught should be carefully avoided by raising the feet above its level, or by sitting out of the current from the door to the fire. Thick boots, especially boots with cork soles, woollen stockings, and gaiters of cloth or leather protect the feet and legs from cold; and warmth to the back of the neck may be afforded by a muffler or high collar. Another aid of great importance in cases of dyspepsia is an apron of chamois leather and flannel or a belt round the abdomen, which should be worn in such a way as to give both warmth and support; support is especially needful in cases where the abdominal muscles are lax and the belly pendulous.

The commonest kind of belt is either flannel or knitted woollen; but the silk scarf known under the name of "cummerbund" (or "camarband" as it is now spelt) is even more comfortable. It should be long enough to go three times round the body, and it can be put on with any degree of tightness. In cases of floating kidney the belt may be provided with a pad or truss [*vide* arts. "Enteroptosis" and "Movable Kidney"].

It is important, as far as possible, to avoid *fatigue* before meals and to get rid of all *worry* or thoughts about business during meals. Some people damage their digestion by walking from their work with the notion of getting an appetite. The extra labour caused by this finishes up the patient already exhausted by his daily work, and lessens the digestive powers still more. Twenty minutes' rest at least, after getting home, is a useful restorative; and is advisable for dyspeptics, especially for patients at or above middle age. On the other hand, if the occupation be of a harassing and anxious kind, it is sometimes useful for the patient to walk home instead of driving in hope of getting rid of his anxiety and worry by exercise. If rest can be taken on arrival the disadvantage caused by the extra bodily work in such cases may be more than compensated by the relief to the mind.

After the meal is over, rest is required both for body and mind; and active exertion, either bodily or mental, is injurious; it is advisable to rest half an hour or more if possible. During this time pleasant conversation or light reading or a pipe may divert the thoughts from care.

The *medicines* that have been used in the treatment of dyspepsia are almost innumerable; but they may be divided into a few classes: (i.) those which stimulate the secretion and movements of the stomach; (ii.) those which by their local action have a sedative action on the stomach; (iii.) those which act upon the general nervous system; (iv.) those which supply digestive material; (v.) those which lessen abnormal decomposition; and (vi.) those which aid in elimination. Among those which stimulate the stomach directly one of the best is bicarbonate of soda in ten or fifteen-grain doses. This should be given fifteen, twenty, or thirty minutes before meals; and it may, I think, be combined advantageously with some bitter, such as calumba, quassia, gentian, or camomile. Another exceedingly useful stimulant is rhubarb.

An old-fashioned plan was to make the patient chew a stick of rhubarb, in which way he got a solution of its active principles in the alkaline saliva; but many persons dislike this way, and prefer the rhubarb taken along with an alkali before meals. A very good remedy is the compound rhubarb powder of the British Pharmacopœia—long known as "Gregory's Powder"—in doses of five or ten grains in a cachet with a mouthful of water twenty minutes before meals. Another very useful mixture is the following, which is much used at St. Bartholomew's Hospital:—℞ Sodii bicarbonat. gr. x., Tinct. gentian. co. ♏ xxx., Spt. chloroform. ♏ x., Infus. rhei ℥ss., Aq. menth. pip. ad ℥j. M. F. hst. mitte doses viii. Sig. Two tablespoonfuls to be taken three times a day twenty minutes before meals. I have already made a comparison between

itching of the skin and irritation of the stomach; and chrysophanic acid, so useful in skin diseases, appears also to have a most useful effect in disease of the stomach.

One of the best combinations that I know is one which was a great favourite of the late Dr. Warburton Begbie; its effects were so good that he used to call it the Pulvis mirabilis:—℞ Bismuth subnit. vel bismuth carb. gr. v., Sodii bicarb. gr. v., Pulv. rhei gr. i., Pulv. nux. vom. gr. ss., Pulv. cinnamon co. gr. iss. M. The powder to be dispensed in cachet; two cachets to be taken with a mouthful of water three times a day twenty minutes before meals. This powder may also be given in cachets containing 10 grains each, and the quantities of the several ingredients may be increased or diminished; so that if the quantity of rhubarb in the above formula cause the bowels to move too freely it may be lessened as required.

A great deal has been written about the uselessness of bitters, and perhaps their utility has been exaggerated; nevertheless in many cases they certainly seem to be productive of great benefit. In cases of atonic dyspepsia, such as we see ordinarily in hospital patients who complain of windy spasm and present a certain definite group of symptoms, quassia and iron before meals appear frequently to be more beneficial than alkalies. The group of symptoms consists in a pale flabby tongue, furred on the dorsum, and marked with teeth at the edges; pain in the epigastrium striking through between the blade bones; much wind in the stomach; flushes of heat; black specks before the eyes, and pain at the top of the head. If constipation be present, some sulphate of magnesia in the morning greatly assists the action of the iron and bitter.

In irritable dyspepsia bismuth is one of our most useful remedies; it may be given in the form either of a solution or of a powder. Some patients object to a powder, and prefer the liquor bismuthi, which may be given in doses of half a drachm to a drachm with aromatic spirit of ammonia and some carminative water. The subnitrate of bismuth does not go very well with bicarbonate of soda, as decomposition occurs with the formation of carbonate of bismuth, nitrate of soda, and evolution of carbonic acid gas, which, if the quantity of bismuth be large, may be so great as either to blow the cork out of the bottle or even to burst the bottle itself. In books on materia medica it is the fashion to recommend the carbonate or subnitrate of bismuth to be suspended by means of mucilage of tragacanth or compound powder; but many patients dislike this thick draught, and prefer the powder simply shaken up in the vehicle. I have already given a formula for bismuth in a cachet with soda. When much pain is associated with taking of food, twenty to thirty minims of the compound tincture of camphor may be added to the bismuth mixture with great advantage. When there is great pain and acidity after meals, it may be relieved by dissolving a teaspoonful of bicarbonate of soda in a little water, and sipping this with a salt-spoon until the pain is relieved; this is, of course, but a palliative measure, yet it gives considerable relief at the time, and (although it is often said to destroy

the coats of the stomach) I have known cases where the practice had been continued for years without any apparent injury to the patient.

Among the drugs which assist the function of digestion through the nervous system, the foremost place must be accorded to nux vomica and strychnine; and ten minims of the tincture of nux vomica, or five of the liquor strychninæ, are most useful adjuncts to antidyspeptic remedies in most cases of feeble digestion. Among those substances which are used to supply digesting material the most important are hydrochloric acid and pepsin; to these perhaps rennet should be added. A dose of ten minims of dilute hydrochloric acid or of nitro-hydrochloric acid appears to help digestion in many cases where the acid only is deficient. It is frequently given with the bitter shortly before meals, as then it seems to stimulate the appetite; but it may be given also immediately after meals, either alone, with tincture of nux vomica, or with pepsin. In some cases rennet appears to be deficient in the gastric juice. The exact use of this ferment in ordinary digestion has not yet been ascertained; but that it has some important function I am convinced, for in making some experiments a good many years ago on the digestive power of various preparations of pepsin, I found one which contained hardly any pepsin but a good deal of rennet; yet, contrary to my expectation, it proved to be of considerable benefit when administered to some of my patients.

Amongst the remedies which tend to prevent fermentation, one of the best is phenol, which may be given in the form of a pill, half a grain to a grain with every meal. Creasote has a similar action, and sulpho-carbolate of soda in ten-grain doses is also a very efficient remedy. For preventing decomposition in the intestines and flatulent distension, besides the remedies already mentioned salicylate of bismuth, with salol or with salicylic acid, is often of great use. The accumulation of fæcal matter in the intestine must be prevented and the bowels kept regular by means of purgatives [*vide* "Constipation," p. 696]. The evacuation of the liver is also a most important matter. The mode of action of mercury upon the liver has not yet been fully ascertained; but there can be no doubt whatever that a dose of blue pill or calomel at night, followed by a saline purgative in the morning, is of the greatest use in relieving many of the symptoms of dyspepsia. In some patients mercurials followed by a saline cause great depression; but this depression may be relieved, without bringing back the dyspeptic condition, by a small glass of champagne or by two tablespoonfuls of brandy in a glass of soda water.

The pains to which I have alluded as probably dependent upon adhesion of the intestine, and occurring in gouty or rheumatic subjects, are best treated by salicylate of soda and salicylate of bismuth with warmth to the stomach and support by means of a belt. Although they may be increased by irritating articles of diet, such as nuts, figs, and like indigestible substances, yet diet does not exercise nearly so much influence over them as exposure to cold, indulgence in acid wines, or in any other excess that usually brings on gout or rheumatism.

T. LAUDER BRUNTON.

REFERENCES

1. ALLBUTT, T. CLIFFORD. *Visceral Neuroses.* Lond. 1884.—2. ALT. "Untersuchungen über die Ausscheidung des Schlangengiftes durch den Magen," *Münchener med. Wochensch.* No. 41, 1892.—3. *Idem.* "Toxalbumine in dem Erbrochenen von Cholerakranken," *Deutsch. med. Woch.* 1892, No. 42.—4. BEAUMONT. *Physiology of Digestion.* Burlington, 1847.—5. BERNARD, CLAUDE. *L'Œuvre de Claude Bernard, Table alphabétique et analytique des œuvres de Claude Bernard.* Paris, 1881. Heading "Digestion."—6. BRUNTON, LAUDER. *Disorders of Digestion.* London, Macmillan and Co.—7. *Idem.* "Snake Venom and its Antidotes," *Brit. Med. Journ.* 3rd Jan. 1891.—8. BLONDLOT. *Traité analytique de Digestion.* Paris and Nancy, 1843.—9. EWALD. *Lectures on Digestion,* New Sydenham Soc. London, 1891 (contains copious bibliography).—10. GAMGEE. *Physiological Chemistry of the Animal Body,* vol. ii. London and New York, Macmillan and Co. 1893.—11. KUHNE. *Handbuch d. physiol. Chemie.* —12. MACFADYEN, NENCKI, and SIEBER. "Chemical Processes in small Intestine in Man," *Journ. of Anat. and Physiol.,* vol. XXV.—13. THOROWGOOD. "Dyspepsia of Liquids," *Lancet,* 1872, vol. i. p. 215.—14. *Vide* also lists of literature in *Index Catalogue of the Surgeon-General's Library,* vol. iii. Washington, 1882, and Neale's *Medical Digest,* London, 1891, with Appendix, 1895.

T. L. B.

DYSPEPSIA IN CHILDHOOD

Disorders of digestion occurring in children below the age of puberty differ in several important particulars from those encountered at a later age. In the first place, the deficient development of the stomach in early life necessitates the greater part of the digestive functions being carried out by the intestine; consequently any failure of assimilation of the food usually manifests itself by symptoms which are intestinal rather than gastric. In the second place, children are very susceptible to catarrhal affections of the gastro-intestinal tract, the symptoms of which are apt to be obscured by phenomena arising from reflex irritation of the nervous system. Chronic inflammation of the stomach, on the other hand, which constitutes one of the commonest causes of dyspepsia in adult life, is rarely met with in young children. Finally, it may be noticed that functional disorders of the stomach associated with an abnormal secretion of gastric juice (Hyperacidity, Hypersecretion) are very rare below the age of puberty.

A. GASTRIC DYSPEPSIA.—1. **Weak digestion.**—This name is given to a functional disorder of the digestive organs characterised by failure of the secretory and motor powers of the stomach and intestine.

Etiology.—In some cases a weak habit of digestion occurs as a primary complaint, but in the vast majority it ensues upon the damage inflicted upon the mucous membrane of the alimentary canal by an attack of catarrhal inflammation. In 38 per cent of the cases of primary dyspepsia which have come under my own care, either one of the parents, or other children in the family, had suffered in a similar manner.

Predisposition to the complaint is more often transmitted through the mother than the father, and is more common in girls than in boys. The children of neurotic parents are particularly apt to fall victims to the

disorder, as are also the offspring of persons who have married late in life. Among the exciting causes of weak digestion, irregularity of feeding and over-indulgence in rich or indigestible substances rank first. In rarer instances the disorder can be traced to overcrowding, deficient ventilation, septic states of the mouth arising from stomatitis or carious teeth, fæcal accumulations in the lower bowel, a neglected hernia, or excessive masturbation. When the complaint is due to antecedent inflammation of the alimentary tract it usually begins between the ages of two and five, and frequently persists throughout life. Occasionally the disease dates from an attack of some infectious disorder, such as scarlatina, measles, small-pox, or pertussis; the symptoms first becoming noticeable during the period of convalescence.

Symptoms.—Pain after food is one of the most prominent features of the complaint. It usually begins shortly after a meal, and is chiefly experienced between the shoulders or behind the lower end of the sterum. Occasionally the skin over the lower ribs on the left side feels bruised and tender to the touch. Flatulent eructations occur after each meal, and may be excited even by a draught of water or a cup of tea. Towards evening the stomach and intestines often become distended with gas, and the pressure which they exert upon the diaphragm gives rise to an inspiratory form of dyspnœa and palpitation. Nausea is a frequent symptom of the complaint, and may persist for many days or even weeks, being temporarily relieved by food and stimulants. The appetite is usually variable, being at one time ravenous and almost insatiable; while at another it can be easily appeased or is altogether absent. The tongue is large, pale, flabby, and often indented along its margins by the teeth. The bowels are usually sluggish in their action, and the stools hard and pale, or putty-like in consistence, but attacks of diarrhœa are apt to supervene from time to time, when the motions not infrequently consist almost entirely of mucus. The pulse is soft, small, and during the periods of digestion abnormally slow. The urine is diminished in quantity, neutral or alkaline in reaction, and deposits phosphates on standing. Vomiting, except as the result of an unusually copious meal, is an inconstant feature of the complaint. Some children, however, habitually vomit solid food, such as meat and fish, whenever they are permitted to partake of it. Although actual emaciation does not usually accompany this condition of feeble digestive power, the child fails to thrive, and becomes dull, spiritless, and disinclined to the least physical or mental exertion. Many patients suffer from extreme languor and drowsiness after meals, or invariably sigh, yawn, or hiccup during the course of the afternoon. Occasionally at these times a strange confusion of ideas may be observed, or the child may stammer or become partially aphasic. In other cases it exhibits various choreic movements, such as nodding the head, blinking the eyes, or incessantly twitching the extremities. In some cases severe frontal headache, which comes on before breakfast and persists until the afternoon, is the chief source of complaint. Among the secondary phenomena of the disease attacks of asthma, palpitation, giddi-

ness, faintness, and irritable skin eruptions are the most important. The asthmatic attacks (asthma dyspepticum) appear suddenly after meals, and are characterised by urgent dyspnœa, cyanosis, and a slow or irregular pulse. These symptoms rapidly subside after vomiting has taken place. Among the cutaneous affections strophulus and urticaria are the most common. The former is especially apt to show itself after indulgence in such articles as porridge, strawberries, fish, almonds, pork and cucumber; while the latter appears chiefly at night-time upon the back, buttocks, and thighs. When the contents of the stomach are examined after a test meal the total acidity is found to be diminished, and the percentage of hydrochloric acid considerably below the normal. Owing to the weak state of the wall of the stomach the period of gastric digestion is much delayed, and fermentation of the stagnant food is apt to occur.

Treatment.—The general principles of treatment are identical with those which are observed in the atonic dyspepsia of later life. The clothing should be warm, and regular exercise must be taken every day in the open air. Excess of fluid with the meals must be avoided, and each mouthful of food must be well masticated before being swallowed. Green vegetables should be given sparingly, and only a small quantity of good potato allowed with the mid-day meal. No food should be permitted after six o'clock in the evening. The first indication for medical treatment is the due regulation of the bowels. When a complete evacuation has been secured by means of castor oil, or an enema, a small dose of cascara combined with maltine may be given twice a day after meals, or some simple aperient may be prescribed each night. As soon as the bowels act regularly the administration of gastric antiseptics may be begun; of these carbolic acid and resorcine are the most suitable and may be given, with bicarbonate of sodium, two or three times a day, about two hours after food. When the abdominal distension and flatulence have quite subsided measures should be taken to stimulate the functions of the stomach. In young children the mixture of rhubarb and soda administered before the meals is usually of great value; but at a later age hydrochloric acid and pepsin are to be preferred. All tonic remedies must be used with caution owing to their tendency to excite gastric catarrh. However, when the gastric symptoms have quite subsided, a careful trial may be made of the milder preparations of iron and cod-liver oil; but in all cases a dose of mercury and chalk or blue pill should be given once or twice a week during the continuance of the tonic treatment.

2. Acute gastric catarrh.—*Etiology.*—Gastric catarrh of an acute or subacute character may occur either as a primary disease, or as a complication of some other and graver condition. The *primary* variety is extremely common in feeble and debilitated subjects, and its inception is favoured by such constitutional diseases as scrofula, rickets, and congenital syphilis. Under such conditions an attack is often directly excited by exposure to wet and cold, especially during the spring and autumn months; but more commonly the cause is to be found in the

administration of unduly rich or indigestible foods. Lastly, it may be mentioned that acute gastro-intestinal catarrh often appears in an epidemic form in localities where typhoid fever is prevalent, and appears to be due to the use of contaminated milk or drinking-water. Gastric catarrh of *secondary* origin is much less common in children than in adults. It chiefly occurs during the early stages of such specific fevers as scarlatina, erysipelas, and influenza; or as the result of chronic diseases of the heart, lungs, liver or kidneys. Occasionally the disease is accompanied by the formation of a false membrane, as in the form of gastritis that sometimes complicates diphtheria.

Clinical varieties and their symptoms.—Owing to the numerous ways in which gastric catarrh may arise, and to the uniformity in the general symptoms of the disease, whatever its exciting cause may be, it is found expedient to divide the complaint into two principal varieties, called respectively the "febrile" and the "non-febrile," according as the temperature of the body is raised or not. (*a*) Febrile gastric catarrh, sometimes known as gastric fever, is a very common complaint in strumous children between three and twelve years of age. It usually begins with shivering, headache, and pains in the limbs; but occasionally its onset is accompanied by convulsions. The temperature rises rapidly and may attain 103° F. by evening, when the child is apt to become slightly delirious. Vomiting is an inconstant symptom, but in some cases repeated emesis occurs, with the ejection of bile and mucus. The bowels are usually confined, but if the catarrhal affection spreads to the mucous membrane of the intestine, the child may have colicky pains in the abdomen accompanied by a watery and offensive diarrhœa. When the duodenum is inflamed jaundice may result. The tongue at first is thickly coated upon the dorsum, while its tip and edges are clean and red. In severe instances the organ becomes dry and fissured, the lips parched and cracked, and sordes appear on the teeth. The appetite is completely lost, and thirst is excessive. Frontal headache, restlessness, and mental depression continue throughout the attack. Sleep is disturbed, and nocturnal delirium may be present. The pulse is quick, weak, and compressible, and the urine is high-coloured, and passed in diminished quantity. The fever is markedly remittent in character, the temperature rising to 101°-103° F. at night, and falling to 99°-100° F. in the morning. It attains its maximum about the third day, after which it gradually declines, becoming intermittent, and finally subnormal. Occasionally the disease ends by a veritable crisis. As a rule an attack lasts from seven to ten days; but if the stomach is irritated by the administration of stimulants or tonics, it may continue for a much longer period. When the disease occurs in an epidemic form the temperature often remains high for ten days or a fortnight, and abdominal pain with diarrhœa is usually present. It subsides in a gradual manner, and may be followed by one or more relapses. The disorder is distinguished from typhoid by its sudden onset and shorter course, and by the absence of tympanites, enlarged spleen, and of the characteristic eruption.

(*b*) Non-febrile gastric catarrh. — There are three clinical varieties of this disease which are worthy of special notice, namely, acute indigestion, subacute gastric catarrh, and recurrent gastric catarrh.

(*a*) Acute indigestion, or as it is often called "embarras gastrique," is most frequent after the age of five years. The symptoms begin suddenly within a few hours of a meal, or during the night; they consist of pain or discomfort at the epigastrium, flatulence, nausea and an excessive flow of saliva. After a variable period of time vomiting occurs, and the stomach rejects the greater portion of the last meal in an undigested state. Although the evacuation of the stomach usually affords relief, the nausea soon returns, and may be accompanied by a sense of faintness or by violent palpitation. The pulse is weak, fluttering, and often abnormally slow; the face is pale, the eyes are sunken and surrounded by dark rings, and the forehead and extremities are bathed in perspiration. These symptoms culminate in violent retching and vomiting, which recur at short intervals, and end in the expulsion of small quantities of bile-stained mucus. The appetite falls into complete abeyance, and any attempt to partake of food or even water only aggravates the retching. In some cases the irritant contents of the stomach find their way into the intestine and set up acute diarrhœa. When improvement sets in the attacks of emesis gradually become less frequent, and finally the child falls into a heavy sleep from which it awakes free from nausea. In rare cases an attack of acute indigestion is ushered in by aphasia, asthma, or a series of epileptiform convulsions, which subside immediately the stomach rejects its noxious contents. The disorder usually runs its course in twenty-four to forty-eight hours, but it is apt to be followed by general malaise, headache, or laryngeal catarrh.

(*β*) Subacute catarrh of the stomach, popularly known as "biliousness," is a very common complaint between the ages of four and sixteen. The subjects of this disorder suffer from chronic depression of spirits, and are listless and apathetic in their movements. They usually present a pale or sallow complexion, with dark lines beneath the eyes, and a slight icteric tinge in the conjunctivæ. In some instances the speech is hesitating or stammering, while in others the patient acquires a habit of frowning, winking the eyes, or making sudden grimaces. The temper is uncertain and peevish, while at night the sleep is disturbed by horrible dreams. In every instance constant and severe headache is a marked feature of the complaint. The pain is usually confined to the frontal region, but it sometimes radiates along the supraorbital nerves. It is most severe in the early morning, or after the mid-day meal, and is increased by active movements of the head, by stooping the body, and by the use of alcohol. Epigastric discomfort is experienced after meals, and towards evening the intestines become distended with gas. Nausea is a common symptom, but vomiting only occurs at intervals or after an unusually full meal. The appetite is much diminished, and the child often expresses the utmost loathing for fat and sweet articles of diet. In the early morning the tongue is usually coated with a yellowish-white

fur, but towards mid-day it may become clean and abnormally moist. Occasionally it is the seat of a symmetrical form of superficial ulceration due to exfoliation of the epithelium. Heartburn and eructations of fœtid gas are a source of frequent complaint after meals, and the patient often yawns, gasps, or suffers from hiccoughs or "goose-skin" during the periods of digestion. The bowels are confined, and the stools clay-coloured and extremely offensive. The pulse is small and feeble, the hands and feet perspire and exhale an unpleasant smell, and attacks of syncope are apt to occur from time to time. The duration of the disease is uncertain. When appropriately treated it usually comes to an end in a few days, but when neglected it may be prolonged for many weeks.

(γ) *Recurrent gastric catarrh.*—This disorder, which has been variously described by the terms "periodic," "fitful," and "cyclical" vomiting, is characterised by severe attacks of emesis which exhibit a tendency to periodic recurrence. The complaint usually begins between the ages of two and five, and according to my experience is more common in boys than in girls. An attack is often precipitated by exposure to cold, a change of diet, or physical or mental fatigue.

Although the attacks exhibit a certain periodicity they do not recur at regular intervals; for at one time several weeks or months may elapse between the consecutive attacks, while at another the disorder may recur once or twice a week. The most severe and obstinate cases are usually those in which the first symptoms date from the convalescent period of measles or typhoid. With the approach of puberty the incidence of the disease becomes less and less frequent, and finally it disappears altogether. In its clinical aspect this variety of gastric catarrh does not materially differ from acute indigestion. As a rule nausea and vomiting are the first symptoms to attract attention, but occasionally severe headache comes on before the emesis. The first effect of the vomiting is to rid the stomach of the remains of the last meal, the constituents of which show few signs of digestion. Severe retching and vomiting recur at short intervals, and the ejecta soon come to consist entirely of thick mucus, or even of bilious fluid. During the course of the disorder the tongue presents a coating of white fur, the bowels are confined, the urine scanty, and the face pale and anxious. The temperature of the body is subnormal. The duration of the complaint seldom exceeds twenty-four hours. Recovery is usually rapid, but when the attacks recur at short intervals the child becomes thin, anæmic, and irritable, and presents the symptoms of weak digestion.

Treatment.—In all inflammatory affections of the stomach and bowels rest in the recumbent posture is an imperative necessity. If the abdominal pain be severe the epigastric region should be covered with a large linseed poultice, or repeatedly fomented with hot flannels. When convalescence has set in the child may be sent to some inland health resort, like Malvern or Ilkley, but sea-air should be avoided. As long as the vomiting continues no attempt should be made to administer food by the mouth, but small pieces of ice may be sucked to relieve the

thirst. When the more urgent symptoms have subsided, a cautious trial can be made of small doses of milk and soda-water, cold bovril, veal or chicken broth. In the febrile form of the disorder a liquid diet should be strictly enforced until the temperature begins to intermit, after which time milk puddings, bread and milk, or toast may be allowed. To prevent a recurrence of the complaint the diet should be strictly regulated to the exclusion of those articles of food that are prone to undergo fermentation in the stomach. Indulgence in sweets and cakes between meals must be prohibited, and the child should be taught to spend a certain time over each meal, and to chew each mouthful of food well before swallowing it. If the case is seen at the beginning of an attack, the stomach should be emptied by means of a full dose of ipecacuanha wine or sulphate of zinc; but if emesis has already occurred the child may be encouraged to drink one or two tumblerfuls of warm water. As soon as the organ has been completely evacuated a few grains of calomel or blue pill may be administered, and followed after a few hours by a full dose of effervescing magnesia or sulphate of sodium. Castor oil is also a valuable remedy at this stage of the complaint, but is apt to excite vomiting. Should the retching continue severe, and signs of exhaustion manifest themselves, it may be necessary to administer a minute dose of morphine, or a sedative enema. In the febrile complaint, where the gastric symptoms are seldom of much importance, repeated doses of calomel, with the occasional use of an effervescing alkaline mixture, are usually sufficient. In the recurrent form of gastric catarrh a dose of the sulphate and carbonate of magnesia every morning before breakfast seldom fails to effect a cure.

B. INTESTINAL DYSPEPSIA.—In addition to the various forms of intestinal indigestion which accompany inflammation of the bowel, and diseases of the liver and pancreas, children are apt to suffer from a curious array of symptoms which appear to depend upon a functional disorder of the large intestine (colonic dyspepsia). Most of the subjects of this disorder either belong to a tuberculous family or present some obvious manifestations of scrofula. The complaint usually begins between the ages of five and ten, and is slightly more frequent in girls than boys.

Symptoms.—Spasmodic pain in the abdomen is the principal symptom of the disease. It usually begins suddenly during the evening or in the early morning before breakfast; or perhaps about mid-day. Sometimes an attack is excited by physical fatigue, or by the use of indigestible foods. The pain is usually referred to the umbilical region, and often travels across the abdomen toward the left. Occasionally it is chiefly felt in the region of the hepatic or splenic flexure of the colon. It may prove severe from the very outset, but it generally grows more and more intense until a maximum is reached, and afterwards gradually declines. During the painful crisis the face is pale and covered with sweat, and the child may roll about the floor or bed and scream with agony. Pressure over the

abdomen usually affords relief. The appetite is extremely capricious, and a marked distaste is shown for fat and sweets. Acid drinks and lemon juice, on the other hand, are much liked. Thirst is a constant symptom, and is apt to be excessive during the night. Constipation exists in about 70 per cent of the cases, but occasionally a lienteric form of diarrhœa is present, the bowels being moved immediately after each meal. Nausea and vomiting are seldom complained of, and the tongue is clean, red, and moist. The victims of this disorder remain thin and anæmic for years, and are prone to gastric or intestinal catarrh from slight causes. After the age of puberty the various symptoms usually subside, but the tendency to constipation continues, and not infrequently an obstinate form of atonic dyspepsia persists for years.

Treatment.—All articles which contain a large percentage of indigestible material must be avoided, hence green vegetables should be given sparingly or withheld altogether. White fish, chicken, and tender meat are to be preferred to the coarser or richer varieties, and all condiments, curries, and highly-spiced foods should be forbidden; milk and cocoa may be given freely, but coffee and strong tea should be prohibited. The meals must be taken at regular hours, and a basin of bread and milk may be allowed when the child retires to bed. The first point that requires attention in the medicinal treatment of the disease is the regulation of the bowels. This is most readily effected by the administration of the liquid extract of cascara sagrada combined with extract of malt immediately after the morning and evening meals. Occasionally small doses of aloine and nux vomica answer well; or some simple laxative, such as the confection of senna and sulphur, may be employed with advantage. When the bowels are moved at each meal a small dose of morphine in an aromatic mixture should be given before the food. As soon as the bowels have been brought into a satisfactory state, one of the bland preparations of iron combined with belladonna will be found of the greatest benefit in preventing a recurrence of the abdominal attacks. Cod-liver oil should not be given until the intestinal pain has quite subsided.

W. SOLTAU FENWICK.

REFERENCES

1. ASHBY and WRIGHT. *Diseases of Children*, 3rd edit. 1896.—2. BAGINSKY. *Die Verdauungskrankheiten der Kinder*, 1884.—3. BEDNAR. *Die Krankheiten der Neugeborenen*, 1850.—4. CLARK, Sir JAMES. *Pulmonary Consumption*, 1835.—5. EINHORN. *Diseases of the Stomach*, 1896.—6. ESCHERICH. *Die Darmbacterien des Säuglings*, 1886.—7. FENWICK, SOLTAU. *Disorders of Digestion in Infancy and Childhood*, 1897.—8. *Idem.* "Dyspepsia of Strumous Children," *Clin. Journal*, Oct. 1894.—9. FENWICK, SAMUEL. *Diseases of the Stomach*, 1869.—10. FOX, WILSON. *Diseases of the Stomach*, 1872.—11. HEURCH. *Diseases of Children*, vol. i. 1889.—12. HOLT. *Diseases of Children*, 1896.—13. ROTCH. *Lectures on Diseases of Infants*, 1896.—14. TODD. Art. "Indigestion," *Cyclopædia of Pract. Medicine*, 1833, vol. ii.—15. ZWEIFEL. *Untersuchungen über den Verdauungsapparat des Neugeborenen*, 1874.

W. S. F.

GASTRITIS

ACUTE GASTRITIS. — SYN.: *Acute gastric catarrh.* — Inflammation of the stomach is divided, according to its course, into acute and chronic; according to its pathology, into mechanical, catarrhal, toxic, symptomatic, diphtheritic, and phlegmonous.

Etiology.—Inflammation of the stomach, like inflammation of any other organ, may be due to mechanical injury, thermal or chemical irritants, or invasion by living organisms either animal or vegetable. Amongst the mechanical causes is to be reckoned, in the first place, excess in food, as when people eat too heartily after a long fast, or are tempted by palatable food and especially by unwonted delicacies; a second cause is an injurious quality of food, which may be either too rough, hard and indigestible, or may be undergoing such changes of fermentation or decomposition as lead to the formation of powerful irritants.

Next come articles of drink, and more especially alcoholic liquors. These may irritate the stomach either (i.) by the amount of alcohol they contain, or (ii.) by the acid which is either originally present in some of them, as for example in certain wines, or is formed from the alcohol in the stomach by acetic fermentation. In many cases the effect of excessive food and excessive alcoholic indulgence are combined to produce acute gastric catarrh.

Poisons, such as acids, alkalies, or salts of the heavy metals, all cause acute inflammation of the stomach; and amongst the mineral poisons one of the most common and powerful is arsenic. Certain organic poisons act as powerful irritants to the stomach, and amongst these may be reckoned some of the products of albuminous decomposition. Some of them may act, either (i.) by direct introduction into the stomach, or (ii.) by excretion into the stomach by its mucous membrane after their formation in the intestine, and elsewhere, and subsequent absorption into the circulation. Antimony when injected into a vein causes vomiting, in part at any rate by its direct action on the stomach; and not by affecting the central nervous system as formerly supposed. It is carried by the blood to the stomach, there excreted, and subsequently exerts a local action on the stomach itself. Many organic substances are excreted in a similar way. Cobra venom was found by Sir Joseph Fayrer and myself to be a powerful local irritant when applied to the stomach of a frog. It seemed to me probable that it would also be excreted by the gastric mucous membrane, and this was found by Alt to be really the case.

Some toxalbumins are excreted by the stomach in cases of cholera also, and have been found by Alt in the vomited matter. The vomiting

which indicates irritation of the stomach at the beginning of many acute diseases is not improbably due to the excretion of toxalbumins or albumoses; and the same is probably the case in the vomiting of patients suffering from renal disease. It is probable, therefore, though not yet certain, that the vomiting symptomatic of gastric irritation in many infective diseases is really of toxic origin.

All the symptoms of gastric catarrh frequently occur after great emotion, and especially after distressing emotions, such as anxiety, sorrow, fright, or anger. It is impossible to say how far these symptoms are due to the direct action of the nervous system on the stomach, and how far to its indirect action in altering the processes of digestion and giving rise to the formation of more or less poisonous products which subsequently act as gastric irritants. The same may be said of external cold as a cause of acute gastric catarrh. In many persons, general exposure to cold, or sitting with wet feet, will bring on gastric catarrh, just as in others it would cause catarrhal inflammation of the trachea and bronchi.

Catarrh of the stomach not infrequently follows catarrh of the respiratory passages. In some people it occurs as the respiratory catarrh is passing off, and in popular language the cold in the chest is said to have been carried off by the stomach and bowels. In some cases the swallowing of mucus from the respiratory passages, and especially from the naso-pharynx, gives rise to gastric catarrh; and it is not infrequent in phthisical patients who swallow the sputum instead of expectorating it.

Inflammation of the stomach may occur from the presence of animal parasites, such as the weevils of cheese, and the larvæ of insects.

Gastric catarrh occurs also in association with organic disease of the stomach, such as stenosis of the pylorus, whether simple or malignant; and it also accompanies malignant disease of other parts of the stomach.

Symptoms.—In acute gastritis, when caused by powerful poisons, there is generally severe pain in the epigastrium with great tenderness and violent vomiting, the vomited matters consisting first of the contents of the stomach, afterwards of mucus, frequently of bile, and finally of mucus tinged with blood, or even of blood more or less pure. As the poison may be completely ejected from the stomach without passing into the intestines, gastric inflammation may occur without inflammation of the intestines; but if this be not the case, and the intestines also become implicated, pain and tenderness extend over the whole abdomen, and diarrhœa more or less violent usually occurs. Even when the inflammation is limited to the stomach the circulation is usually much affected. The face becomes exceedingly pale and pinched, the surface is cold, there is abundant cold perspiration, and frequently also a profuse flow of saliva. The pulse may be quick or slow, but is generally feeble. In the case of a powerful poison, these symptoms are apt to remain more or less persistently for several days.

When the irritation is less powerful, as is the case where the stomach has been simply overloaded with food or drink, or where the food or drink has been of an unsuitable nature, the mere evacuation of the stomach by vomiting gives much relief, and the patient may feel almost well; although a certain amount of weakness and lassitude usually remains behind. If the vomiting is at all violent the tenderness in the stomach may continue for some days; and sometimes a soreness, which is probably due to straining of the œsophagus, is felt all down the chest behind the sternum for a day or two. This rapid disappearance of symptoms usually occurs, however, when the irritant has been applied for a short time only, and when the mucous membrane of the stomach has not been much inflamed. When the mucous membrane itself is inflamed certain symptoms remain after the evacuation of the strong irritant to which the inflammation is due.

These symptoms may also appear more gradually in consequence of the continuous action of a slighter irritant; in which case, instead of beginning with violent vomiting and pain, which subside to a certain extent after the evacuation of the stomach, the symptoms go on gradually increasing for days or weeks, until they may culminate in great pain and vomiting such as has just been described. These symptoms usually are a thickly furred tongue and loss of appetite, frequently accompanied by a good deal of thirst. Not only is there no desire for food, but eating may bring on a feeling of nausea, and nausea may arise at the mere thought of food. Frequently, however, although ordinary food is distasteful, there is a craving for strong-tasting and savoury things, such as salt fish, pickles, curry, Worcestershire sauce; and occasionally the appetite may be somewhat craving rather than deficient; but after a little food has been taken this craving appetite is usually soon satisfied and turns to loathing.

The stomach is tender on pressure, and, being distended with gas, is often prominent in the epigastrium. Gaseous eructations are frequent, and may be accompanied by sour fluid so acrid as to burn the throat, to set the teeth on edge, and to give rise to much discomfort at the lower end of the ensiform cartilage—at the point corresponding to the junction of the œsophagus with the stomach. In some cases hiccough is a persistent trouble, and there is a feeling of weight and pain in the epigastrium which seems to pass through between the shoulder-blades. The bowels are costive unless the catarrhal condition extends to the intestines as well. This is to be anticipated as very little food is taken or absorbed. The urine is generally scanty, high-coloured, and deposits a brick-dust sediment. Generally also there is a feeling of weight in the head, and headache, frontal, temporal or occipital, is frequent; sometimes patients complain that the pain feels like a ball inside their head. There is difficulty in concentrating the thoughts; all exertion, mental or bodily, is distasteful, and the patient is very low-spirited and not infrequently irritable. The temperature is often normal, but in some cases it rises as high as 102° or 103°; this rise is usually preceded by a rigor.

Pathology.—Our knowledge of the slighter stages of acute gastric catarrh is derived almost entirely from the observations of Dr. Beaumont on the stomach of a young Canadian voyager, Alexis St. Martin. In consequence of a severe wound in his side from a discharge of duck-shot, his stomach became adherent to the abdominal wall, and an opening existed in it, usually covered by a fold of mucous membrane which, when pushed back, revealed the interior of the stomach. Dr. Beaumont describes the inner coating of the stomach, in its natural and healthy state, as being of a light or pale pink colour, varying in its hues according to its full or empty state. "It is of a soft or velvet-like appearance, and is constantly covered with a very thin, transparent, viscid mucus lining the whole interior of the organ. Immediately beneath the mucous coat, and apparently incorporated with the villous membrane, appear small spherical or oval-shaped granular bodies, from which the mucous fluid appears to be secreted." With a thin whitish fur on the tongue and a rather craving appetite, "several red spots and patches, which were tender and irritable, appeared over the inner surface of the mucous membrane." When the countenance was sallow, the tongue covered with a thin white coat and the appetite failing, the stomach presented several deep red patches on its inner coat. When sick headache was present, with pain and uneasiness of the stomach, general debility, lassitude, a depressed pulse, dry skin, coated tongue, and costiveness, the stomach presented numerous white spots, or pustules resembling coagulated lymph spread over its inner surface. After indulgence in spirits—with a thin, yellowish-brown fur on the tongue and uneasy sensation and tenderness at the pit of the stomach, vertigo, dimness and yellowishness of vision on stooping down and rising again, and a sallow countenance, but no general discomfort nor failure of appetite—the mucous membrane of the stomach was found to present an erythematous appearance and livid spots, from the surface of which exuded small drops of grumous blood, numerous patches of aphthæ, and a thick coating of mucus; the gastric juice was also mixed with thick, ropy mucus or muco-purulent matter, slightly tinged with blood, resembling the discharge from the bowels in dysentery. The tenacious secretion from a stomach in such a condition is found by microscopical examination to consist of mucus with leucocytes and epithelial cells undergoing mucoid degeneration. The cylindrical epithelium of the tubules undergoes mucous degeneration and desquamation; and in such cases microscopical examination of the mucous membrane itself shows the peptic cells to be loosened and granular, and the epithelial cells at the mouth of the tubules to be soft, swollen, filled with mucus, and desquamating. The blood-vessels are distended, the tissues are filled with leucocytes, the submucous tissue is somewhat œdematous, and there may be slight hæmorrhage.

Diagnosis.—The symptoms of acute gastric catarrh are so distinctive that usually little doubt can exist regarding the nature of the attack; but it is not always easy to say whether it may not be due to the pre-

sence of toxins or to the invasion of pathogenetic organisms, such as typhoid bacilli, which may affect other organs as well.

If the attack be not accompanied by a febrile temperature, and there is a clear history of some dietetic error or exposure to a chill, the probability is that the symptoms are due to acute catarrh only; but if the temperature be high the attack may denote the onset of some infective disease, and a diagnosis can only be arrived at by watching the case for some days. Gastric catarrh is most likely to be confounded with commencing enteric fever, but in the latter disease the rise of temperature is less abrupt, the remissions are more marked, and the duration of the fever is more prolonged.

Cases of acute gastric catarrh usually end in recovery in one or two days, although they may last for as long as a week or perhaps even for a fortnight. Such prolonged cases may well be mistaken for typhoid fever; but in addition to the diagnostic points already mentioned, the spots are absent and the spleen is not enlarged.

When the attack sets in with severe headache and delirium, it may be mistaken, especially in children, for meningitis, or for the beginning of measles, or of scarlet fever; but the course of the disease and the absence of the rash after its proper time enable a diagnosis to be made.

The treatment of acute gastric catarrh consists first in removing all irritant substances from the stomach; and, secondly, in soothing the inflamed organ. The stomach naturally tends to eject any irritating substance present in its cavity; but, after the main portion of its contents has been ejected by vomiting, small quantities of exceedingly bitter bile mixed with mucus may remain and give rise to constant nausea and retching. In order to remove this it is advisable to administer several tumblerfuls of lukewarm or of warm water, and, after this has been vomited, to repeat the process several times; thus all irritant substances are washed out of the stomach, and after this has been done two or three times the vomiting will often cease. Occasionally the first draught of two or three tumblers of water will so dilute the contents of the stomach that the irritation is no longer strong enough to produce vomiting. In such a case it is better to tickle the fauces with a feather, so that the water may be vomited and the stomach cleansed; or the gastric syphon may be used: but unless the patient is accustomed to the use of this instrument the plan of swallowing hot water and tickling the fauces is generally preferred. In mild cases this treatment is all that is required, and after a few hours' rest the stomach is all right again; but for at least a day afterwards the diet should be very light, consisting chiefly of milk and farinaceous food. When the attack is more severe and the catarrh has affected the bowels also, as shown by a tendency to diarrhœa and pain not confined to the gastric region but extending over the abdomen, half an ounce of castor oil with 8 or 10 minims of laudanum should be given; so as to clear out any irritant from the intestine. If, after the oil has acted, the pain or nausea or vomiting still persist, or if there be any yellowness of the con-

junctivæ, half a grain of calomel may be given every half hour till 5 grains have been administered; or 5 to 10 grains may be given at night, and next morning a saline purgative, such as a glass of aperient mineral water or some effervescent sulphate of magnesia, sulphate of soda, or phosphate of soda.

The best food is simply milk diluted with one-fourth to one-half its volume of soda water; unless the bowels be loose, when lime-water should be substituted. This dilute nourishment may be given in quantities of 5 or 6 ounces every two hours; but should it be rejected by the stomach, it is well to let the patient take nothing but a little iced water and swallow small pieces of ice until the acute irritation has subsided. If there be any fear of failure of strength, the nutrition may be maintained by nutritive suppositories or nutritive enemas. After the irritant matters have been evacuated from the stomach, one of the best sedatives is bismuth, which may be advantageously given according to the following formulas:—℞ Bismuth. carb., sodii bicarb. āā gr. x., spt. chloroformi ♏ x., aquæ menthæ pip., vel aq. cinnamomi, vel aq. flor. aurant. ℥j. M. Ft. hst. ante cibos sumend.

If there be much pain, 5 minims of tincture of opium or liquid extract of opium may be added to each dose; and if the vomiting be severe 5 minims of hydrocyanic acid may be added, either with or without opium, as the case seems to demand.

Should the bowels be loose some chalk may be added to each dose, or two drachms to an ounce of chalk mixture may be given either with the bismuth mixture or after each loose motion.

If the bismuth tend to constipate the bowels two fluid drachms or more of liquor magnesiæ carbonatis may be added to each dose.

Counter-irritation to the epigastrium by a mustard poultice or mustard leaf tends to relieve both pain and vomiting; and a full warm bath is often very soothing and useful both in the case of children and of adults.

CHRONIC GASTRITIS.—SYN.: *Chronic gastric catarrh, Chronic dyspepsia.* —**Etiology.**—The causes of chronic catarrh are the same as those of acute catarrh; and attacks of acute catarrh, especially if frequently repeated, pass into a chronic condition.

The irritation of the stomach which produces chronic rather than acute catarrh is usually less intense in degree, and frequently repeated.

In acute catarrh the very violence of the irritation causes ejection of the irritant, while in chronic catarrh, the irritation being less severe, the irritant remains in the stomach, producing perhaps no more than discomfort at the time, but gradually altering the mucous membrane and even the muscular coats. One of the most common causes of common gastric catarrh is free indulgence either in acids or substances which yield acids in the stomach. Thus among the peasantry of Southern Europe sour wine is a frequent cause of gastric catarrh; and all wines, even those which taste sweet, are strongly acid to litmus-paper. Spirit,

when taken in a concentrated form, will act as a powerful irritant; when more dilute, it frequently undergoes acetic fermentation, and thus acts as an acid. Sugar, especially when taken in a concentrated and soluble form, is apt to give rise to acidity; while starches, being less soluble, are generally passed through the stomach without undergoing such a change, although if the stomach be dilated they also may undergo fermentation. Fats are apt to undergo change also and to yield butyric acid, which is one of the most irritating of all acids to the stomach. But people frequently forget that a rancid fat, which would make them sick if taken by itself, does not lose its irritating properties when it is mixed up with flour and made into pastry. Pastry is often blamed as a fruitful cause of gastric catarrh; but frequently the blame is not due to the pastry itself, but to the kind of fat with which it is made: thus pastry made at home with the best butter may sometimes be taken with impunity, when pastry from the pastry-cook would surely disagree. Another cause is eating too quickly; another is drinking much liquid with meals. Both of these practices tend to interfere with the rapid digestion of food, and thus to allow time for fermentative processes to go on. Retention of food in the stomach with consequent fermentation and chronic gastritis occurs when the stomach is dilated, in consequence either of atony or of pyloric obstruction due to cicatricial contraction or to new growths. Malignant disease in other parts of the stomach may directly give rise to catarrh.

Obstructed circulation through the stomach tends to induce chronic catarrh; thus it is frequently found associated with conditions which obstruct the gastric circulation; such as cirrhosis of the liver, phthisis, and mitral disease, obstructive or regurgitant.

An altered condition of the blood, such as anæmia, chlorosis, renal disease and gout, may lead to chronic catarrh.

Pathology.—It has already been mentioned that in acute inflammation mucoid degeneration of the cells in the gastric glands and mucous degeneration and desquamation of the epithelium occur. In chronic catarrh the same processes take place, their long continuance leading to wasting of the mucous membrane with increased formation of connective tissue and a tendency to the formation of cysts in the tubules. Sometimes the atrophy occurs fairly evenly over the whole surface of the stomach; but at other times cystic degeneration occurs to a large extent in certain parts, so that the mucous membrane, instead of being smooth and thin, rises in folds and knobs, or polypi. For this reason it has been described as "gastritis polyposa." In the cases just mentioned the change chiefly affects the mucous membrane, and there is no very marked increase in the connective tissue. The connective tissue in some cases of chronic disease of the stomach is very little altered; in others it undergoes great increase. The increase may take place both in the upper and lower layers of the mucous membrane, and in some cases the increase of connective tissue is so great that the walls of the stomach are greatly thickened and the stomach itself diminished in size. To this condition the name of "cirrhosis" has been given.

Symptoms.—As in acute catarrh, chronic irritative conditions of the stomach may be accompanied by a craving appetite, or by loss of appetite. The tongue is usually coated; there is weight, distension, uneasiness, oppression or pain in the epigastrium; a good deal of flatulence; sometimes a disagreeable taste in the mouth; frequently tenderness on pressure. The pharynx is often congested; there may be a tendency to hawk and spit, and sometimes there is a tickling, irritating cough, which may come in severe paroxysms. Eructation of sour fluid and pain at the pit of the stomach are also present, such as have been described already under dyspepsia. Headache, generally frontal, is often complained of, and there is indisposition to any exertion mental or bodily, depression of spirits, and often irritability of temper. In some cases there is distinct giddiness or palpitation.

Diagnosis.—Three forms of chronic gastric catarrh have been described by Ewald, namely, simple, mucous, and atrophic. In the first two hydrochloric acid is diminished, but the normal ferments are present, and the secretion of mucus is scanty; in the second form the mucus is abundant. In the third form both hydrochloric acid and the characteristic ferments of the stomach are absent. The methods of ascertaining these conditions have already been given under the heading of "Pathology of Digestion."

Primary atrophy of the stomach has been described by Fenwick. Its symptoms are progressive weakness and anæmia, with a sallow colour almost exactly like that of pernicious anæmia. This condition comes on very gradually without any apparent cause, generally attacks persons past middle life, and on post-mortem examination the gastric mucous membrane has been found pale, generally thin, and adherent to the submucous tissue. The gastric glands are firmly united and distended with cells and molecular matter. At a later stage the tubules disappear completely, or sometimes a number of flask-shaped bodies loaded with cells remain at the bases of the tubules, while in the rest of the mucous membrane no glandular structure can be recognised.

The diagnosis between chronic gastric catarrh and malignant disease is often quite impossible. If the symptoms persist in spite of treatment, but the patient is under forty, the most probable diagnosis is chronic gastric catarrh; if the symptoms persist in spite of treatment in a patient over the age of forty the presence of malignant disease is exceedingly probable. In a case of long-standing chronic catarrh, it is not until the presence of a tumour becomes evident that the diagnosis of malignant disease can be made with certainty. Malignant disease indeed may come on in persons who have long suffered from the symptoms of chronic gastric catarrh. One of my patients had been accustomed to wash out his stomach for three years, and not until after the lapse of this time did it become evident that he was suffering from malignant disease. In another patient, who suffered for fourteen years from dyspepsia, the stomach had been dilated for at least nine years, but only five months before death did a tumour become evident.

In order to distinguish cases of chronic atrophy of the stomach from

pernicious anæmia, the attention should be directed to the presence or absence of the gastric ferments and of hydrochloric acid in the stomach, to the absence of minute, very large, or distorted corpuscles in the blood, and to the presence of amines and iron in the urine.

Treatment.—The first essential in the treatment of chronic gastric catarrh is to remove the sources of the irritation which brought it on. The general dietetic treatment has already been given under "Dyspepsia," and I may sum it up shortly by saying the patient must avoid alcohol, sugar, and condiments, eat fat sparingly, chew slowly and masticate thoroughly. Liquids and solids should not be taken together; and, if necessary, farinaceous and proteid foods must be taken at different times. The body should be kept warm, the mind easy, and the bowels open. In cases of dilatation, great relief is afforded by washing out the stomach. This is best done in the morning, so that the long night's rest may allow as much food as possible to be absorbed, and the waste of washing out the stomach be as small as possible. For further information on this subject the reader is referred to the article on "Gastrectasis." Water just slightly coloured with permanganate of potash may be used instead of plain water; or two to five grains of boric acid may be added to every ounce of the water.

T. LAUDER BRUNTON.

REFERENCES

1. BRUNTON and FAYRER. *Roy. Soc. Proc.* vol. xxi. p. 358 and vol. xxii. p. 68.—2. ALT. "Unters. über die Ausscheidung des sub-cut. injicert. Morphium durch den Magen," *Berl. klin. Woch.* xxvi. 25, 1889.—3. *Idem.* "Untersuchungen über Ausscheidung des Schlangengiftes durch den Magen," *Münchener med. Woch.* xxxix. 41, 1892.—4. FENWICK, SAM. *Morbid States of the Stomach, etc.* Lond. 1868.—5. LEUBE, W. Ziemssen's *Handb. der spec. Path.*—6. EWALD, C. A. *Klinik der Verdauungskrank.* Berlin, 1890-93.—7. LEO. *Diagnosis der Krankheiten der Verdauungsorgane.* Berlin, 1890.—8. PICK, A. *Vorl. üb. Magen- u. Darmkrankheiten.* Wien, 1895.—9. OSER. "Magen Krankheiten," *Eulenburg's Realencyklopädie,* vol. xii.—10. ROSENHEIM. *Path. u. Therapie der Krank. der Verdauungsapparat.* Wien, 1894.—11. FLEINER. "Erfahr über die Therapie der Magenkrank.," *Volkmann's Vorh.* No. 103.—12. WEGELE. *D. phys. u. medikam Behandlung der Magen- u. Darmerkrank.* Theil ii. Jena, 1895.—13. DEBOVE and RENAULT. *Ulcère de l'estomac.* Paris, 1892.—14. MATHIEU. *Thérapie des malad. de l'estomac.* Paris, 1895.—15. OSER. "Neurosen des Magens," *Wien Klinik,* xi. Nos. 5 and 6, 1885.—16. LEUBE. "Nerv. Dyspepsie," *D. Archiv klin. Med.* Band 23, p. 98, 1879.—17. DEBOVE and REMOND. *Traité des maladies de l'estomac,* 1883.—18. PENZOLDT. "Allgemeine Behandlung der Magen- u. Darmkrankheiten," *Handbuch der specillen Therapie innern Krankheiten,* 1895, Bd. iv.; contains very complete bibliography up to date.—19. LEVEN. *Estomac et Cerveau.* Paris, 1884.

T. L. B.

PHLEGMONOUS GASTRITIS.—This is a severe inflammation passing on to purulent infiltration of the stomach wall. Its main incidence is on the submucous coat, though it usually affects the other coats also to some extent; thus, as well as by its severity and gravity, it is distinguished from ordinary inflammations of that organ which mainly involve the mucous coat, and are generally spoken of as catarrhs [*vide* previous sections]. Fortunately it is a rare disease. The total number of cases recorded up to the present time number only about 85; of these

11 have occurred in this country. It is met with in two well-marked forms: (A) the diffuse, and (B) the circumscribed; in the former the suppurative process is more or less widely and diffusely spread throughout the stomach wall, in the latter it forms a definite collection of pus in the gastric wall—in other words, an abscess. In three of the cases a combination of both forms was present.

A. THE DIFFUSE FORM occurs with nearly twice the frequency of the other.

Pathological anatomy.—The stomach wall is invariably thickened, sometimes greatly so; even eight or nine times that of the normal. The thickening may be general and uniform but is more frequently

FIG. 16.

local, the pyloric region being its usual seat. The capacity of the stomach, as a whole, is sometimes considerably enlarged, often unaltered, rarely diminished. (*a*) *The mucous coat* is most frequently swollen and hyperæmic, sometimes it even presents hæmorrhages. In a fair number of cases, though swollen, it is pale throughout; in a few it seems to be normal. The morbid appearance may be continued for a short distance into the duodenum. It is not a little remarkable that the continuity of the surface is rarely broken. Slight ulcerations have been met with in two or three cases; and actual perforations, extending into the submucous coat and allowing of pus being expressed on squeezing, have been observed in five cases only. Microscopically, the chief change is an abundant round-celled infiltration between the gastric glands; there

may be dilatation of the superficial blood-vessels also, and cloudy swelling of the secreting cells. The muscularis mucosæ may be normal, or irregularly swollen in places and infiltrated with round cells (Fig. 16). The changes are indicative of an acute pathological process. (*b*) *The submucous coat.* — The thickening is here extreme, though its extent varies in different cases, and often in different parts of the same case. It is white or pale yellowish white in colour, and of a soft, diffluent, or even fluid consistence; it is rarely firm and solid. It is usually continuous over the affected part, though it may be interrupted, and may show specially swollen foci in places. It is generally sharply bounded

Fig. 17.

by the pylorus; but occasionally it extends into the duodenum for a short distance. In the same way it may infiltrate the walls of the lower end of the œsophagus. An œdematously swollen zone is usually seen surrounding the purulently infiltrated part. Microscopically, immense numbers of small round cells are seen everywhere throughout its thickness, sometimes so numerous as completely to obscure the natural structure. Fibrin threads may also be present. The blood-vessels are dilated and the tissues of their coats are swollen (Figs. 16, 17). The disease appears to begin in the deeper layers of the submucosa. Micro-organisms in great abundance, especially streptococci, have been found in a few (seven) of the more recently recorded cases. (*c*) *The muscular coat.* —It is often unaltered, often swollen and œdematous, less frequently infiltrated with pus or a mixture of pus and serum; and in a considerable

number of cases it is more or less completely destroyed in whole or part, thus allowing the pus to reach the serous coat. Microscopically, a plentiful cellular invasion of the lymph spaces and connective tissue strands between the muscle bundles may be seen in cases where to the naked eye the muscle appears normal; and the muscle fibres may be swollen, granular, or fatty. (Figs. 17, 18). In other cases the muscle fibres are more or less completely necrosed. Micro-organisms may be seen throughout its whole thickness. (*d*) *The serous and subserous coat.*—They are unaffected in a great many cases, but an œdematous swelling or a purulent infiltration of the latter and a dimming of the gloss of the former may be seen. In

FIG. 18

some cases the inflammation of the serous coat is well marked, and exhibits the various degrees of a local peritonitis. The same micro-organisms may be found here as in the other coats. (*e*) *Complications.*—General peritonitis is by far the most frequent; it occurred in half the cases. Among other lesions pericarditis, pleurisy, abscess of the liver and parotitis have been observed.

Etiology.—(*a*) *Sex.*—The disease is much more frequent in men than in women. Oser, in a total of 27 cases, gives the proportion at 8 to 1; while in a total of 47 cases I have found it to be about 5 to 1.

(*b*) *Age.*—It is most frequent in early adult and middle life. I have been able to ascertain the age in 43 cases, and find 3 between 10 and 20, 13 between 20 and 30, 7 between 30 and 40, 9 between 40 and 50, 7 between 50 and 60, 3 between 60 and 70, and 1 between 70 and 80. The youngest was 11 and the oldest 70.

(*c*) *Alcoholic excess.*—This factor has been thought to play an important part in the production of the disease, as it was present in nearly one-half of the cases collected by some observers. However, this proportion is undoubtedly too great; one-fifth or one-sixth would be more correct: moreover, when we compare the rarity of the disease with the frequency of alcoholic excess, we cannot place much reliance upon it.

(*d*) *Dietetic errors.*—In a certain proportion of cases the disease has followed immediately after a meal, so that the food has been suspected. Two of the patients habitually ate to excess after periods of fasting or spare feeding; but in none of them could a more direct connection of this kind be traced.

(*e*) *Injury.*—There is a history of a previous injury to the epigastric regions in two of the cases only.

(*f*) *Blood-poisoning.*—The great majority of cases are primary, and are thus called "idiopathic." A few are secondary, having followed upon some form of injury or operation in six cases, upon puerperal fever in one, typhus fever in one, gastric cancer in two, and gastric ulcer in one. These are called "metastatic" cases. In the idiopathic forms the infection must come from the side of the stomach or from that of the blood. In the former case it must arise in (α) the food introduced from without; (β) the secretions which act upon it; (γ) the poisons which may be produced by the action of the normal or abnormal ferments or accidental gastric contents. Ordinary alimentary substances can hardly be thus harmful, even if we take into account individual idiosyncrasies concerning shell-fish, mackerel, and the like. The ordinary gastric secretions, namely hydrochloric acid and pepsin, have been experimented with. Bouveret and others assert that the former, and Bergmann and others that the latter, is injurious when injected into rabbits, and will kill them. Trypsin may get back into the stomach; and it has been shown by Pawlow and others to produce a local necrosis and inflammation, which may be hæmorrhagic, but not suppurative. The third division includes a variety of conditions which as yet are imperfectly known to us. Stricker and Kocslakoff found that pus sometimes appeared in the submucous coat of the stomach after injection of a small dose of ammonia and water; and Pilliet produced a phlegmonous gastritis by the injection of caustics (sulphuric acid and croton oil essences) which differed from that of man in being more hæmorrhagic, less purulent, and free from micro-organisms. We can hardly suppose, however, that substances similar to these are formed within the stomach. On the other hand, the stomach usually contains a great number of bacteria, and the entrance of one or other of these into its walls might lead to the characteristic results. The sudden onset, rapid course, and pathological appearances presented by the disease all favour this explanation of its nature. It is not necessary that the organism should gain an entrance locally through an abrasion of the mucous membrane; it is more probable indeed that it does so by the blood; for we rarely find evidence of such absorption in cases, such as gastric ulcer or neoplasm, where an obvious means of ingress is present.

It will be remembered that very few cases of phlegmonous gastritis show any breach of surface in the mucous coat; further, the anatomical characters presented by the openings, if present, suggest that they follow the purulent formations in the submucous coat rather than precede them. To elucidate this point I examined microscopically a number of cases in which the stomach wall was acutely thickened, coexistently with some abrasion of the mucous coat; and I very rarely found any evidence of septic absorption. A leucocytic invasion of the submucous coat was sometimes seen; but it was noteworthy that the invading cells were chiefly the lymphocytes seen in simple inflammations, and not the multinucleated leucocytes seen in septic cases; moreover, organisms were absent. The stomach has a remarkably active power of repair; losses of substance tend to heal by granulation and cicatrisation, and do not readily allow of septic absorption. A certain parallel may be drawn between phlegmonous gastritis and the so-called idiopathic cellulo-cutaneous or cellular forms of erysipelas. The cause of the latter is the streptococcus so abundantly found in the lymphatic vessels and spaces bounding the affected area; but we do not know how or why it entered. We know that it is more prone to attack patients who are the subjects of general debility, of chronic alcoholism, or of chronic renal or hepatic disease, or patients with a hereditary or acquired predisposition; but there our knowledge ceases. Nevertheless, we do not say of the origin of idiopathic erysipelas as we do of phlegmonous gastritis, that it is entirely unknown; yet the two diseases have much in common. Indeed it is possible that the same organism, the streptococcus, is the immediate cause of both maladies. It will be remembered that suppuration is usually seen in the more virulent forms of erysipelas. Phlegmonous gastritis might thus be regarded as an erysipelas of the stomach; Virchow indeed likened the disease to a carbuncle. We are on less certain ground when we attempt to state the conditions which favour the development of the streptococcus in the stomach wall. Alcohol in excess would appear to have a certain influence, perhaps even more than it has in the production of an idiopathic erysipelas, because the stomach, in addition to its increased vulnerability as a part of a deteriorated organism, suffers from the local irritation set up by its contents. Dietetic errors may act in much the same way, excessive loading of the stomach may render it sluggish, and favour the deposition and multiplication of organisms in the submucous coat. Such suggestions are, however, but speculations.

Symptoms.—The sudden onset, the epigastric pain, and the vomiting are among the most frequent and trustworthy symptoms; but as peritonitis is so frequent a complication we must endeavour to distinguish the symptoms due to it from those proper to the gastritis. It is present in a large proportion of the cases, probably in one-half. In cases from which it was absent the leading symptoms were epigastric pain, vomiting, fever, restlessness, anxiety, delirium, collapse and coma; and these it will be useful to examine separately.

(i.) *Pain* was present in about 75 per cent of the cases. It varied from a mere sense of discomfort or weight in the epigastrium to violent pains of a cutting or boring kind. It generally appeared with more or less suddenness on the first day of the illness; but occasionally not till the second, third, or fourth day. It usually increased in severity, and kept pretty steady, being but rarely paroxysmal and rarely subsiding for any length of time. It was nearly always localised at first to the region of the epigastrium, spreading either slowly or suddenly to the rest of the abdomen with the onset of general peritonitis. The early pain is probably due to the irritation of the nerves so plentifully present in the submucous coat, and to its steady increase on the extension of this irritation and the subsequent implication of the serous surface. The pain is increased by pressure: Leube says it is not; but further experience has shown that the cases of Claus, Raynaud, and Stewart, on which he founded his statement, were exceptional in this respect. Occasionally pain has been elicited by pressure when it was previously absent.

(ii.) *An increased sense of resistance.*—This was generally present also in the epigastric region at an early stage; probably in many cases before the local peritonitis had appeared. Later the upper part of the abdomen frequently became prominent and swollen, and this local meteorism soon became general.

(iii.) *Vomiting* is a symptom which seldom fails. Leube says "it was absent in only one of the cases recorded; and even then there was a disposition to emesis." A more extended observation shows that it was absent in six cases; namely, in both the cases occurring in cancerous stomachs (Gläser and Mintz), in Macleod's and Mascarel's idiopathic cases, in Manoury's case of puerperal fever, and practically so in Gläser's case of ulcer, in which vomiting occurred once only, on the second day. Vomiting was frequently preceded by loss of appetite, irritation, and nausea, generally of short duration as in most cases the vomiting itself set in on the first day. In one case of five and a half days' duration it appeared on the second day; in another, of eight days' duration, it began on the fourth day; and in another, of nine days' duration, it did not come on till near the end. It was frequently repeated, sometimes almost continuous, without any abatement until death; but in many cases it ceased in a day or two, sometimes altogether; sometimes it came on afresh as peritonitis set in. The vomited matters at first are watery, and contain alimentary matter and mucus; but in almost all cases they soon become tinged with bile, becoming yellow or sometimes greenish. In no case was pus recorded in the vomited matters; and this is remarkable, as we remember that sieve-like openings, large enough to allow of the escape of pus into the stomach, were present in five cases. Probably its escape into the stomach took place too slowly or in quantities too small to attract attention. It is unlikely that it became altered by the gastric juice, for probably this secretion is largely if not entirely in abeyance. (iv.) *Jaundice*, generally to a very slight degree, was present in about 16 per cent of the cases. (v.) *Temperature.*—In most cases there was some

fever, the range varying from 100° to 104° F., but in a few cases, in some of which peritonitis was present, there was no perceptible rise of temperature. (vi.) *The pulse* is generally full, strong, and at first but slightly accelerated. Later it becomes more rapid, feebler, and often irregular. (vii.) *The bowels.*—Diarrhœa was present in a certain number of cases, constipation in others; sometimes constipation preceded diarrhœa. (viii.) *The mouth* is generally dry, the tongue is dry and covered with a white fur, and thirst and anorexia are present. (ix.) *Mental phenomena.*—These varied somewhat, but restlessness and anxiety are generally pronounced from the very beginning of the disease, and increase as it progresses. The patient looks and feels severely ill, and often has the aspect of the "typhoid" condition. Wandering speech and delirium are frequent, and death usually follows from coma or collapse.

Diagnosis.—The sudden onset, the localised pain increased by pressure, the sense of increased resistance over the region of the stomach, the bilious vomiting and fever, all point to a gastritis. The rapidly increasing severity of the symptoms, the gravity of the case, and the early onset of peritonitis, may suggest its phlegmonous character. Chvostek claims to have diagnosed one of his cases during life, but it is obvious that, as a rule, we cannot hope to do more than guess at the diagnosis.

Prognosis.—This is as grave as it can be. The course of the disease is rapid, and its duration brief. The mean duration is about six and a half days. The shortest course was one day, and the longest seven weeks. There is no authentic record of a recovery, if we except Dittrich's two preparations in the museum at Erlangen which are regarded by Raynaud, Leube, and others as evidence that recovery is possible.

Treatment can be but symptomatic. All alimentation should be by the rectum.

B. THE CIRCUMSCRIBED FORM.—It is not necessary to treat of this form with the same fulness as the former, as we need only discuss the characteristics peculiar to it.

Pathological anatomy.—The whole stomach is not so much altered as in the diffuse form. The abscess is the most prominent change. It varies in size from a bean to thrice the size of a man's closed fist. It may be single or multiple, and it occupies a definitely circumscribed position in the submucous coat. The mucous coat is generally raised inwards over the tumour, and is thinned to a degree which varies with the size of the abscess. It may be pale or reddened, intact, or perforated by one or more apertures. Some authors think that a perforating gastric ulcer may arise in this way. The muscular coat is generally thinned also, or sometimes softened and destroyed, so that the abscess approaches the serous coat. A purulent peritonitis may thus arise; but it may appear before this and without an actual extension of the pus outwards through the muscular coat. The abscess may rupture into the peritoneal or pleural cavities, into the lumen of the stomach, or even through the anterior

abdominal wall. The rest of the stomach may show little or no change. Secondary abscesses may appear in the liver or other organs.

Etiology.—We distinguish between the idiopathic and the metastatic varieties; and the latter occurs probably with a relatively greater frequency than in the diffuse form. Cases are recorded as occurring in the course of puerperal fever, ulcerative endocarditis, and like maladies. Bacteriological observations have not been made; but following the line of argument employed in discussing the diffuse form, we may fairly conclude that it is caused by the lodgment, growth, and development of certain pyogenetic cocci within the submucous coat. In all probability it is not invariably due to the same microbe.

Symptoms.—They closely resemble those of the diffuse form, especially when the case is acute. The localised pain increased by pressure, and the increased resistance in the epigastric region are generally speaking more marked, while the rest of the abdomen is less painful. In the chronic cases the symptoms are less pronounced, the pain, vomiting, irregular fever, loss of appetite and strength with gradual emaciation being the most suggestive. Occasionally all characteristic symptoms were absent. In a few cases a distinct tumour in the epigastrium has been felt during life, which was not always painful on pressure. The vomited matter may contain pus, or pure pus may be vomited in large quantities.

Diagnosis.—In cases where the symptoms are well marked and a palpable tumour appears in the epigastric region, a diagnosis may be made with some probability. It may become more certain on the vomiting of pus. In Callow's case 20 oz. of pure pus were vomited in the presence of the physician, and other like cases have been recorded.

Prognosis.—It is also grave, but several cases of spontaneous recovery after the vomiting of pus have been recorded.

Treatment.—Symptomatic until the presence of a tumour in the epigastric region renders the diagnosis probable. Surgical interference should then be resorted to without delay. If an abscess be found it should be freely drained. To wait for the possible chance of a natural evacuation of the pus into the cavity of the stomach is dangerous, as every hour increases the risk of a fatal peritonitis. If an operation be undertaken in time, its results ought to be more promising than they are in subphrenic abscess and gastric ulcer, the successful issue of which may now be added to the triumphs of modern abdominal surgery.

MEMBRANOUS GASTRITIS.—This condition, which is also spoken of as croupous and diphtheritic gastritis, is a rare disease. It is much more frequently met with in children than in adults.

Pathological anatomy.—The mucous surface of the stomach is covered by a membrane of various thickness, colour and extent. It is sometimes so thin that it is difficult to keep it whole; at other times it forms a thick and consistent layer. The colour varies from a white or whitish gray to a whitish or grayish yellow or even brown tint. Its

usual extent is within narrow limits; it very rarely covers the inner surface from the cardiac to the pyloric end of the organ. It mostly appears in separate patches or elongated strips along the swollen mucous ridges, and usually it is firmly adherent to the mucous membrane beneath, which in turn is usually swollen, reddened and even hæmorrhagic. Generally speaking, it cannot be detached without damage to the surface of the mucous membrane. In a great many cases there is membrane also upon the pharynx or larynx: occasionally it is continuous over the surface of the œsophagus from the mouth to the stomach; but this is a rare occurrence. Usually the membrane stops abruptly at the junction of the pharynx and œsophagus to reappear in the stomach. Occasionally there is a membranous enteritis or colitis also. The microscopic structure of the membrane has been described by Bizzozero, Kundrat, and Smirnow, who agree that it consists mainly of a network of fibrin, containing in its meshes leucocytes, red blood cells, and degenerated epithelial cells. Smirnow was able to distinguish two forms: one a croupous form, consisting mainly of exudative products, and not sending processes downwards into the mouths of the gastric tubules; and the other a diphtheritic form, containing in addition the results of a coagulative necrosis of the superficial epithelial lining of the stomach, and dipping into the tubules. The inflammatory changes in the mucous membrane itself are more intense in this form than in the other, and extend somewhat into the underlying coats. In Dr. John Thomson's case the membrane consisted almost entirely of leucocytes held together by a small amount of fibrin; it contained no epithelial cells. The data afforded by bacteriological examinations are at the present time too scanty to allow of any inference from them. Smirnow found in it both cocci and bacilli, and Wollstein many micrococci; but Thomson found few or none.

Etiology.—The disease is almost always secondary to some other disease. A certain amount of evidence has indeed been brought forward to show that it may be primary, both in children and adults. Niemeyer says that in some cases, in infants, the catarrhal form of inflammation increases to the croupous, but he probably based his opinion upon theoretical considerations rather than upon practical experience; for in the very few cases in which we have had an opportunity of examining the stomach in the primary acute catarrh of children, it has been found swollen and intensely congested, even to a dark red colour, but not covered by a membrane. The evidence in favour of primary occurrence in adults is hardly more convincing. It rests mainly upon the case in a man aged forty-six, reported by Delafield.

The secondary form in children may occur at any age. It has been found in new-born children who have died of pyæmia (Bednar, Orth, etc.) or of deficient nutrition and debility (Parrot). Later in childhood it most frequently follows diphtheria, as shown by Jenner, Seitz, Smirnow, Kundrat, Jones, and others. It has also occurred in cases of scarlatina, measles, cholera infantum, and small-pox, where there was a membrane in the throat also; and a considerable number of cases have been reported

in children dying of tuberculosis. In adults the disease is secondary to diphtheria, typhoid, typhus, puerperal fever, small-pox, ulcerative endocarditis, corrosive poisons, and so forth. Fox has seen it in phthisis, and Wilks both in gouty kidney and hepatic abscess. It is quite possible that the direct causation varies in different cases. They all agree that the condition arises in patients weakened by severe disease, in many of whom there is also a pellicle in the mouth or throat. This pellicle is not always the same. Sometimes it is a simple thrush; sometimes a true diphtheria; sometimes the membranous exudation of scarlatina. The special significance of Löffler's bacillus enables us to distinguish genuine diphtheria from the non-diphtheritic cases, which often simulate it closely. The particular organism present in the membrane in the mouth may readily reach the stomach, and there set up a somewhat similar condition; or it may be even possible for the gastric ferments, by chemical action independent of organisms, to produce the gastritis and the membrane. In diphtheritic cases the condition of the stomach, as of the fauces, is doubtless due to the local action of Löffler's bacillus; and whether we are entitled to distinguish between them and others, which have been called croupous but would be better called fibrinous or exudative, is as yet undetermined. It is at least probable that such a distinction does exist, and that a membranous gastritis, like a membranous enteritis, may exist apart from diphtheritic infection.

Symptoms.—In many cases there are no gastric symptoms of any kind; in others the ordinary symptoms of severe gastritis are present; namely, vomiting, epigastric pain and tenderness, anorexia, thirst, and fever; but in any case the symptoms due to the primary disease are usually so grave as to mask those proper to the gastric condition. Occasionally shreds of the membrane have been coughed up; and in Thomson's case the whole of it, forming a cast of both the cardiac and pyloric ends, was thus brought up. It is probably in such cases only that a diagnosis would be possible.

TOXIC GASTRITIS.—This name signifies the changes produced in the stomach by the ingestion of poisonous doses of various substances, of which the chief are concentrated acids and alkalies, arsenic, alcohol, phosphorus, and certain salts; for example, corrosive sublimate, copper salts, etc.

Pathological anatomy.—The extent and intensity of the changes depend upon the amount, concentration, and kind of the poison. When dilute, they act only upon the mucous coat; when concentrated, they act on all the coats, producing sloughs more or less deep, surrounded by a zone of intense congestion, and accompanied by a sanguineous effusion into the coats and cavity of the stomach. Alkalies generally penetrate more deeply than acids. The poison acts chiefly upon that part of the stomach wall with which it first comes in contact. Sometimes it acts upon this part only, as peristalsis may be abolished, or the poison so quickly diluted by other gastric contents that when distributed

over the remaining surface it causes no further damage. Thus the middle of the greater curvature opposite the œsophagus, or the cardiac or pyloric regions, are often chiefly or exclusively affected. Sometimes both the cardiac and pyloric portions suffer, while intervening parts escape. The pylorus is frequently contracted. In the most severe cases the affected part may be completely dissolved, so as to produce a perforation, usually circular, but varying in size, and with highly congested and hæmorrhagically infiltrated margins; and by this opening the gastric contents escape into the peritoneal cavity. The remaining part of the stomach may show little or no change, although more than half of the organ may have been destroyed. The surrounding organs often show characteristic changes, or an acute peritonitis may be set up. Sometimes there is no actual perforation, but such complete softening of the stomach wall that it tears upon the slightest touch. In less severe cases the mucous coat is intensely red and injected, often presenting sloughs or ulcers, especially upon the summit of its folds, accompanied by hæmorrhagic œdema of the other coats, particularly of the submucosa. The purulent or gangrenous process thus set up may spread for a time, but subside short of perforation, and be counteracted by a healing process. Granulation tissue is thus formed, which results in cicatrisation; very extensive deficiencies may be partly remedied in this way, as the stomach has a great power of repair. The scar tissue contracts greatly; and, though the mucous membrane over it is not regenerated, the resulting defect may be very small. The contraction, however, may lead to great deformities, such as stenosis of the pylorus, hour-glass contraction, and pouching of the stomach. This general summary does not take note of the more intimate changes in colour, and other features, produced in the mucous membrane by the separate poisons; but a few brief references may be made to them.

The corrosives.—Sulphuric acid is the most common of these poisons. It produces a dark red surface with necrosed patches, especially upon the rugæ; these are at first gray or grayish white, later they become dark brown or black, as if charred. This change may be seen over the whole or the greater part of the inner surface of the stomach. The colour will be modified by the amount of food in the stomach at the time, or by the amount of mucus coating its surface. If the coating be thick the parts appear as if smeared with white paint. Dr. Wilks mentions a case, in a woman who lived several days after drinking dilute sulphuric acid, in which the whole mucous membrane, of a bright yellow colour, was found lying loose within the stomach. Perforation is more frequent than with any other acid; it occurs in about one-third of the cases. Nitric acid usually produces a yellow or green staining, and less corrosion than sulphuric. The effects of hydrochloric acid sometimes resemble those of nitric, and sometimes of sulphuric acid. No case of perforation has been put on record. Caustic alkalies and their carbonates produce inflammation, with abrasion, ulceration, and hæmorrhage. The sloughs are softer, more gelatinous, and less regularly

limited. Wilks says they convert the mucous membrane into a tawny pulp, and often perforate the stomach. So far as I can find, however, perforation did not occur in any of the recorded cases; but in one case, in a woman who died a few hours after drinking a 30 per cent solution of caustic potash in mistake for iodide of potassium, the stomach was found to be almost completely dissolved.

The non-metallic irritants.—The salts of the alkalies and alkaline earths may produce intense inflammation of the mucous membrane, with ulceration and black patches resembling gangrene. Potassium nitrate and some of the barium salts have been known to cause perforation, but this accident is very rare. Phosphorus causes inflammation and softening of the mucous membrane with frequent petechiæ and even erosions. Fatty degeneration of the epithelial cells of the surface and glands quickly follows. Iodine causes inflammation and brown discoloration, sometimes with erosion.

The metallic irritants.—Arsenic may cause nothing but a red colour and an injection of the blood-vessels; but when large doses are taken one or more patches are usually formed, varying in size from a sixpenny piece to that of a crown, consisting of mucus or of a tough white, yellowish, or even violet-coloured coagulated lymph, mixed with arsenious acid, firmly fixed to the mucous membrane, with signs of intense inflammation around them. White spots of arsenic are also sometimes found between the rugæ. The stomach contents are usually of a dark brown colour; but sometimes yellow from the partial conversion of the poison into the sulphide. Occasionally the surface of the stomach looks as if it had been coated with yellow paint. Ulceration is rare; perforation and gangrene still rarer. The salts of antimony usually set up intense inflammation only. Corrosive sublimate produces softening, sloughing, and ulceration. The ulcers are often numerous and small, and show a grayish white deposit of mercury on their surface. The copper salts produce much inflammation and ulceration, and a green or sometimes blue colour.

Organic irritants.—Oxalic acid acts mainly as an asthenic, but may act on the mucous membrane of the stomach, producing a pale or highly inflamed rugose appearance, especially towards the pylorus. This coat is shrivelled, easily stripped off, and in some cases is partially and even extensively detached; the vessels are minutely injected with black blood. Perforation is rare. Wilks mentions a case in which after two weeks a gastritis was set up, and the patient vomited a greenish brown fluid; after death the mucous membrane was found to be greenish yellow, injected, and in parts abraded. Among the inebriants alcohol may cause nothing, or an intense dusky red colour and even extravasation. Carbolic acid causes a pale, corrugated, sodden, and partially detached mucous membrane, or a dry surface as if tanned. Cantharides produces intense inflammation, and even gangrene, in patches where the powder has adhered. After ethereal oils or acrid vegetable or animal poisons severe injection or membranous inflammation may be seen.

over the remaining surface it causes no further damage. Thus the middle of the greater curvature opposite the œsophagus, or the cardiac or pyloric regions, are often chiefly or exclusively affected. Sometimes both the cardiac and pyloric portions suffer, while intervening parts escape. The pylorus is frequently contracted. In the most severe cases the affected part may be completely dissolved, so as to produce a perforation, usually circular, but varying in size, and with highly congested and hæmorrhagically infiltrated margins; and by this opening the gastric contents escape into the peritoneal cavity. The remaining part of the stomach may show little or no change, although more than half of the organ may have been destroyed. The surrounding organs often show characteristic changes, or an acute peritonitis may be set up. Sometimes there is no actual perforation, but such complete softening of the stomach wall that it tears upon the slightest touch. In less severe cases the mucous coat is intensely red and injected, often presenting sloughs or ulcers, especially upon the summit of its folds, accompanied by hæmorrhagic œdema of the other coats, particularly of the submucosa. The purulent or gangrenous process thus set up may spread for a time, but subside short of perforation, and be counteracted by a healing process. Granulation tissue is thus formed, which results in cicatrisation; very extensive deficiencies may be partly remedied in this way, as the stomach has a great power of repair. The scar tissue contracts greatly; and, though the mucous membrane over it is not regenerated, the resulting defect may be very small. The contraction, however, may lead to great deformities, such as stenosis of the pylorus, hour-glass contraction, and pouching of the stomach. This general summary does not take note of the more intimate changes in colour, and other features, produced in the mucous membrane by the separate poisons; but a few brief references may be made to them.

The corrosives.—Sulphuric acid is the most common of these poisons. It produces a dark red surface with necrosed patches, especially upon the rugæ; these are at first gray or grayish white, later they become dark brown or black, as if charred. This change may be seen over the whole or the greater part of the inner surface of the stomach. The colour will be modified by the amount of food in the stomach at the time, or by the amount of mucus coating its surface. If the coat be thick the parts appear as if smeared with white paint. Dr. Wi mentions a case, in a woman who lived several days after drin dilute sulphuric acid, in which the whole mucous membrane, of a b yellow colour, was found lying loose within the stomach. Perforati more frequent than with any other acid; it occurs in about one of the cases. Nitric acid usually produces a yellow or green sta and less corrosion than sulphuric. The effects of hydrochloric sometimes resemble those of nitric, and sometimes of sulphuric. No case of perforation has been put on record. Caustic alkalie their carbonates produce inflammation, with abrasion, ulceratio hæmorrhage. The sloughs are softer, more gelatinous, and less re

limited. Wilks says they convert the mucous membrane into a tawny pulp and often perforate the stomach. So far as I can find, however, perforation did not occur in any of the recorded cases; but in one case, in a woman who died a few hours after drinking a 30 per cent. solution of caustic potash in mistake for iodide of potassium, the stomach was found to be almost completely dissolved.

The non-metallic irritants.—The salts of the alkalies and alkaline earths may produce intense inflammation of the mucous membrane, with ulceration and black patches resembling gangrene. Potassium nitrate and some of the barium salts have been known to cause perforation, but this accident is very rare. Phosphorus causes inflammation and softening of the mucous membrane with frequent petechiæ and even erosions. Fatty degeneration of the epithelial cells of the surface and glands quickly follows. Iodine causes inflammation and brown discoloration, sometimes with erosion.

The metallic irritants.—Arsenic may cause nothing [illegible] a red colour and an injection of the blood-vessels; but when large doses are taken one or more patches are usually formed, varying in size from a [illegible] penny piece to that of a crown, consisting of [illegible] of a [illegible] white, yellowish, or even violet-coloured [illegible] with arsenious acid, firmly fixed to the mucous membrane [illegible] inflammation around them. White [illegible] found between the rugæ. The stomach [illegible] brown colour; but sometimes yellow [illegible] poison into the sulphide. Occasionally [illegible] as if it had been coated with yellow [illegible] and gangrene still rarer. The [illegible] inflammation only. Corrosive [illegible] and ulceration. The ulcers are [illegible] grayish white deposit of [illegible] produce much inflammation [illegible] blue colour.

Organic irritants.—Croton [illegible] act on the mucous membrane [illegible] inflamed [illegible] is shriveled, easily scraped off [illegible] extensively detached [illegible] blood. Perforation is rare. [illegible] weeks a [illegible] was [illegible] fluid; after death the [illegible] yellow, [illegible] may cause vomiting, if in [illegible] tion. Corrosive acid causes [illegible] detached mucous membrane [illegible] produces intense inflammation [illegible] powder has adhered, after [illegible] poisons [illegible]

Symptoms.—They are of sudden onset, especially in the case of the corrosives. A sudden penetrating heat is felt in the stomach at the moment when the corrosive reaches it; it soon increases to a vivid burning, excruciating pain in the stomach, œsophagus, mouth and throat. The epigastrium is sensitive to pressure. An intense thirst sets in which no drink can assuage. Swallowing is painful or impossible. Nausea, retching, and vomiting follow, and become more and more frequent, often almost constant. The vomited matters contain food, blood, mucus, flakes of epithelium or of the mucous membrane, and frequently traces of the poison. The abdomen at first is usually retracted, but afterwards it may become distended. There is often much bloody diarrhœa coming on shortly after the vomiting; sometimes constipation and tenesmus. The urine is scanty or suppressed. The face is anxious; the pulse frequent, small, and sometimes irregular; the respirations quickened; the extremities cold, and the skin covered with clammy sweat. The lips and mouth are blistered and shrivelled, and may show spots of a characteristic colour or be cyanosed. The neck and clothes may also show signs of the poison. In fatal cases the patient becomes cyanosed, his respiration is embarrassed, and he sinks into collapse and dies; it may be by suffocation or in convulsions. All these phenomena come on quickly, sometimes in two hours or even less. The vomiting may cease, but the pain becomes generalised; meteorism and the other classical signs of peritonitis appear, and the patient dies in three or four days. If the quantity swallowed be not very great the symptoms may amend in a few days. The patient may suffer from fever, breathlessness, occasional vomiting, indigestion, and gradual emaciation, and die after the lapse of a few weeks or months. He may vomit portions of the mucous membrane of the gullet or stomach. Laboulbène mentions a case in which, fourteen days after poisoning by sulphuric acid, a membrane was vomited measuring 20 cm. long by 12 cm. broad, in which no glands were found. Rather less than half the adult patients recover completely from the corrosive acids, but recovery may be incomplete, the patient being permanently weakened by means of the cicatricial contraction of œsophagus or stomach or by impairment of digestion. In the case of such irritants as phosphorus, arsenic, antimony, corrosive sublimate, the copper salts, and the like, the symptoms are much the same; but the pain and the vomiting do not usually supervene until some hours have elapsed after the swallowing of poison. Death may follow in twelve to twenty-four hours. If it do not, the symptoms of general intoxication due to the particular poison are soon added to those of the gastritis.

Diagnosis.—This is usually easy. If the patient or his friends do not tell the story, an examination of the mouth will show whether a corrosive has been taken, and usually also which it is. If the mouth and throat show nothing, a careful examination of the vomited matters will usually lead to the detection of the poison, and later the symptoms peculiar to each intoxication may appear, and clear up any remaining difficulty.

Treatment.—The first indication is to get rid of the action of the poison, or to counteract it. In the case of irritants which do not act immediately, the stomach-pump or a free syphonage of the stomach may be promptly used, if we see the case early enough. Emetics, such as powdered ipecacuanha root, mustard, or an injection of apomorphine, may be given when the stomach-tube is inadvisable. In the case of the corrosives its use is contra-indicated, and mild stimulants and diluent drinks should be given, such as milk and water—mucilaginous and oily drinks in the case of the acids—with dilute alkaline solutions such as magnesia or chalk and water; or, in the case of the alkalies, with dilute acid solutions such as water and vinegar, or dilute citric acid or lemon juice. These drinks should be frequently repeated for some hours, or even for days. If swallowing be impossible an attempt should be made to introduce a tube beyond the obstruction in the œsophagus, so as to administer the antidote through it. Against phosphorus 40-minim doses of oil of turpentine in mucilage should be given every fifteen minutes for an hour. The oil should be old oil, or French oil. Magnesia, also in mucilage, should be given next day. Against arsenic the hydrated sesquioxide of iron, and the hydrated oxide of magnesia, are to be prescribed; against corrosive sublimate the white of egg. The next indication for treatment is to combat the collapse and the pain by injections of ether, caffein, camphorated oil, friction to the skin, injections of morphia, ice or iced drinks by the mouth, and perhaps leeches to the epigastrium. The subsequent gastritis is to be treated in the usual way.

REFERENCES

Phlegmonous Gastritis.—1. Bouveret and Bouchard. *Traité de pathologie générale*, 1895, tome i. p. 781.—2. Callow, *vide* Auvray, *loc. cit.*—3. Claus. *Beitrag zur Kenntniss d. Magenkrankheiten*, Inaug. Diss. Erlangen, 1857.—4. Gläser. *Berl. klin. Wochnschr.* 1883, Bd. xx. S. 790.—5. Macleod. *The Lancet*, London, 1887, vol. ii. p. 1166.—6. Manoury. *Bull. Soc. Anat. de Paris*, 1842, tome xvii. p. 175.—7. Mascarel. *Ibid.* 1830, tome xv. p. 176.—8. Oser. *Real-Encyclopaedia Magenentzündung*, 1887, Bd. vii. S. 412.—9. Pilliet. *Bull. Soc. Anat. de Paris*, 1893, No. 22, and *Bull. méd. Paris*, Dec. 13, 1893, and Jan. 17, 1894.—10. Stewart, Grainger. *Edin. Med. Journal*, 1868, N.S. vol. xii. p. 732.—11. Stricker and Kocslakoff. *Sitzungsb. d. k. k. Akad. d. Wissensch. Wien.* 1866, vol. iii. p. 781.

For a fuller account of the disease with a record of cases, see paper by the author in *The Edinburgh Hospital Reports*, vol. iv. 1896. The disease is one of considerable antiquity, as it was noticed by Varandæus in 1620, *Tractatus de Morbis Ventriculi*. Cases were recorded in the latter half of the 17th and the beginning of the 18th centuries by Borel, *Opera* 1656; Sand, *Dissertatio de raro Ventriculi Abscessu*, Regiomont, 1701; Vorwaltner, *Eph. Nat. cur.* Dec. 3, Obs. 142; Bonetus, *Sepulchretum sive Anatomia Practica*, lib. iii. Geneva, 1700. These all referred to the circumscribed form. The earliest reference to the diffuse form is by Cruveilhier, *vide* Raynaud, *Bull. Soc. Anat. de Paris*, 1861, tom. vi. pp. 62-93. Among the many papers on the subject, for which see author's monograph above mentioned, are Auvray, *Thèse de Paris*, 1866. Leube, Ziemssen's *Cyclopædia of the Practice of Medicine*, 1877, vol. vii. p. 154. Fagge, *Trans. Path. Soc. London*, 1874-75, vol. xxvi. p. 81. Chvostek, *Wien. med. Presse*, 1877, Nos. 22, 29, vol. xviii. s. 693, and *Wiener med. Blät.* 1881, No. 28, vol. iv. ss. 831, 861, 891, 924, 962. Glax, *Berl. klin. Wochensch.* 1879, vol. xvi. s. 565, and *Deutsche med. Ztg.* Berl. 1884, No. 3. Ackermann, *Virch. Archiv*, 1869, vol. xlv. s. 39. Habershon, *Guy's Hosp. Rep.* London, 1855. Testi, *Ann. univ. di med. e chir. Milano*, Dec. 1883,

pp. 523-547 (with a record of the older and oldest literature of the subject). Hun, *New York Med. Journ.* 1868, vol. viii. p. 18. Mintz, *Deutsches Archiv f. klin. Med.* Leipzig, 1892, Bd. xlix. s. 487. Kelynack, *The Lancet*, London, 1896, March 14.

Membranous Gastritis.—1. BEDNAR. *Krankheiten de Neugeb. und Sauglinge*, 1853. —2. BIZZOZERO. *Medicinische Jahrbucher*, 1866, S. 207, *vide* Smirnow.—3. CHIARI. *Resp. d. k. k. Krankenanst. Rudolph Stiftung in Wien*, 1880.—4. DELAFIELD. *Morbid Anatomy*, p. 140.—5. FOX. *Diseases of the Stomach*, 1872.—6. JENNER. *On Fevers and Diphtheria from* 1849 *to* 1879, p. 498.—7. JONES. *The British Medical Journal*, 1889, i. p. 880.—8. KUNDRAT. Gerhardt's *Handbuch der Kinderkrankheiten*, Bd. iv. S. 388.—9. NIEMEYER. *Text-book of Practical Medicine*, 1873, vol. i. p. 503.—10. ORTH. *Lehrbuch d. speciellen path. Anat.* 1887, iii.—11. PARROT. *Le progrès médical*, 1875, No. 28, p. 393.—12. SEITZ. *Diphtheria and Croup.* Berlin, 1877.—14. SMIRNOW. *Virch. Archiv*, Bd. cxiii. S. 333-356.—14. THOMSON, JOHN. *Archives of Pediatrics*, Philadelphia, April 1895.—15. WILKS. *Pathological Anatomy*, 3rd edition, 1889.—16. WOLLSTEIN. *Archives of Pediatrics*, 1892, p. 489.

Toxic Gastritis.—1. BÖHM. Ziemssen's *Handbk. der speciellen Pathol.* xv.—2. LABOULBÈNE. *Bull. de l'acad. de méd.* 1876, p. 1145.—3. LESSER, A. *Virchow's Archiv*, 1881, lxxiii.—4. ORTH. *Lehrb. der speciellen pathol. Anat.* 1887, iii.—5. WILKS and MOXON. *Pathological Anatomy*, 3rd edition, 1889.

GANGRENE OF THE STOMACH.—**Pathology.**—A gangrenous condition of part of the stomach wall is seen comparatively seldom. It is most frequently met with when the stomach is the seat of a cancerous growth. Every pathologist is familiar with the foul, ragged, and sloughing masses often seen in this disease; they may be present in the floor and margins of the malignant growth and in its vicinity, and may reach a large size. The cancerous growth, by interference with the vessels, deprives the tissues, especially those of the mucous coat, of their nutrition, and they die. The gastric juice may thus act upon these devitalised tissues as it does upon the cancer itself, and cause their partial or complete solution. It is by this process, more frequently than by the extension of the cancer itself, that peritonitis arises in cases of gastric cancer.

Again, gangrenous patches or extensive sloughs are frequently seen after the ingestion of various poisons, notably the mineral acids and alkalies. Sloughs of the mucous membrane have also been met with in a few cases of phlegmonous gastritis, and also in membranous gastritis. Occasionally the floor of an ordinary perforating ulcer shows sloughy tissue. It is quite possible that a gangrenous or at any rate a necrotic process may precede the formation of many such ulcers. Necrosis naturally follows the cutting off of the blood-supply to a portion of the stomach wall, and on the solution of this under the action of the gastric juice the ulcer will arise. The microscopic appearances presented by the margins of an ordinary gastric ulcer, not undergoing healing, tend rather to support this view, for there is none of the vascular or tissue reactions met with in an ordinary ulcer elsewhere. I have met with one case, in a man aged about fifty, in which a gangrenous process was associated with a gastric ulcer. At the lesser curvature, close to the œsophageal end, was an area about the size of a two-shilling piece, partly gangrenous and partly perforated. The perforation was in its upper part; it was about

the size of a sixpence, and its upper margin had clearly defined and clean-cut edges; its lower margin was formed by the peritoneal and shreddy subperitoneal coats. This part gradually got thicker, forming a black sloughing tissue which, at the margin of the area, was generally abruptly marked off from the surrounding healthy mucous membrane. An early local peritonitis, most marked in the region of the ulcer, was also present. Mayer's case, reported also by Leube, in which perforation occurred during life near an old cicatrised ulcer, has many resemblances to this case. The perforation occurred in a black, discoloured, irregularly softened area, the characters of which were not unlike those produced by cadaveric softening. It took place suddenly after the patient had partaken heartily of new beer and black bread. Similar perforations have occurred in the œsophagus (Dittrich, Chiari), and it is probable that in all these cases a hæmorrhagic infiltration of the walls of the organ had first taken place, and was followed by maceration under the action of the gastric juice. Recklinghausen's case, where a gangrenous patch about the size of a shilling followed from a thrombosis of a branch of the coronary artery, may be regarded as corroborating this view. Habershon describes two cases, one in a woman aged thirty-three with pneumonia and paraplegia, and the other in a man aged fifty-one with pneumonia and renal disease, where there were several black sloughs situated in cup-shaped depressions in the mucous membrane. Fox mentions that he has seen sloughs of the mucous membrane in cases of gangrenous pneumonia, and Recklinghausen describes a case of acute tuberculosis in a man whose stomach showed several elevated necrotic patches which contained a fungus believed by him to be their cause. Klebs also met with gangrenous patches in the stomach in a case of acute yellow atrophy of the liver in a woman of twenty, and in a case of gangrenous stomatitis in a child of four. Necessarily rare, however, as such cases undoubtedly are, we may conclude, I think, that severe injury, such as the severe over-distension in Mayer's case, or septic emboli, especially in patients whose strength has been sapped by severe disease, may set up arterial obstruction and consequent infarction, or a direct necrosis. We have, lastly, to determine whether a primary gangrene of the stomach exists apart from the action of corrosives. On theoretical grounds we may suppose that it does. If primary suppuration exist, why not gangrene? Yet very little evidence can be brought forward in support of this opinion. The sloughing patches which occur in the mucous membrane of the stomach in anthrax can hardly be cited, as they exist only along with intestinal lesions; but Klebs has described a case in which the swollen mucous membrane of the stomach showed a few brownish-red patches like partially gangrenous areas. These presented an abundant round-celled exudation, and numerous spore-containing bacilli, varying in length from 5·9 to 11·7 μ; these were found upon the free surface and in the lumen of the gastric glands, frequently separating the cells from the basement membrane. There were no round cells except where the bacilli were present, the latter apparently coming first. Nasse and

Orth describe a somewhat similar case in a man aged sixty who suddenly fell ill and died after vomiting much blood. The diagnosis was gastric ulcer, but the necropsy showed that there were many surface losses in the mucous membrane, and that the gangrenous process had spread deeper than in the last case, having at one place eroded a branch of the coronary artery. Bacilli were present in these lesions also. It would thus appear that a local primary gangrene of the stomach may occur, but that it is among the rarest of pathological curiosities.

Symptoms.—There are no symptoms proper to the gangrenous process, except the appearance of shreds of the mucous membrane in the vomited matter; and this happens chiefly in toxic gastritis. They should be carefully looked for in other cases, however, as I was once able from a microscopic examination of a shred of tissue thus brought up to diagnose a cancer of the stomach in a man under thirty years of age, in whom all the other symptoms were consistent with simple catarrh or ulcer. In most cases the gangrene is entirely overshadowed by the concomitant disease, and in others the signs suggest no more than an ulcer or an acute gastritis.

REFERENCES

1. CHIARI. *Prager med. Wochenschr.* 1884, No. 24.—2. DITTRICH. *Ibid.* 1885, No. 40.—3. FOX. *Diseases of the Stomach*, 1872, p. 115.—4. HABERSHON. *Guy's Hospital Reports*, 1855, 3 s. 1, p. 116.—5. KLEBS. *Allgemeine Pathologie*, 1887, Bd. i. S. 207, and *Virchow's Archiv*, Bd. xxxii. S. 198.—6. LEUBE. Ziemssen's *Cyclopædia of the Practice of Medicine*, 1877, vol. vii. p. 264.—7. MAYER. *Deutsches Archiv für klin. Med.* 1871, Bd. ix. S. 105.—8. NASSE and ORTH. *Virchow's Archiv*, Bd. civ. S. 548.—9. RECKLINGHAUSEN. *Virchow's Archiv*, 1864, Bd. xxx. S. 366.

CIRRHOSIS OF THE STOMACH

In this, almost the rarest of stomach affections, there is marked thickening of the submucosa, accompanied also by thickening in the muscular and serous coats. It has been described by various writers under different synonyms, namely, plastic linitis by Brinton, hypertrophy by Fox, fibroid induration by Handfield Jones, sclerosis by Sneller, cirrhosis, and so on.

Pathological anatomy.—The stomach as a whole is markedly diminished in size, firm, rounded and elastic. In advanced cases it resembles a thick-walled india-rubber tube, blown out at one end (cardiac). It is increased in weight, and when opened does not collapse. When cut it often gives rise to a creaking sound like cartilage, and there is considerable resistance to the passage of the knife through its walls. The cavity is much reduced; in some cases its capacity falls to little more than two ounces of fluid. The thickening of the wall is greatest in the pyloric region, frequently attaining six to eight times the normal mean, and it may even reach the thickness of an inch. It gradually diminishes until the cardiac extremity is reached, where it may be slight or practically absent. In a certain number of cases it involves only the pyloric half of the organ; very rarely, indeed, does it affect the whole organ uniformly.

It is said not to pass beyond the pyloric and œsophageal openings respectively; but in a case of my own this change was found throughout the gastro-intestinal tract from stomach to rectum, excepting the duodenum and upper jejunum. The aperture of the pylorus may or may not be contracted. The colour of the section of the stomach wall is a dull opaque white, and all the coats—mucous, submucous, muscular, and serous—stand out clearly from one another. The chief thickening is in the submucosa; but it may be surpassed by that of the muscular coat. The mucous membrane is usually pale and smooth, or ridged or mammillated, that is, beset with little projections closely studded together; but it is sometimes congested, sometimes excoriated and ulcerated. The microscope usually shows very little thickening, or atrophy, or signs of catarrh. The gastric tubules are rarely separated, bent, or deformed by any interstitial fibrous growth. The cells may be granular or fatty, but in many cases, and in many sites in one case, it may seem very little altered. In my own case there was in places a fairly abundant small round-celled exudation, of a recent nature, irregularly distributed between the tubules; and the secreting cells themselves were smaller than normal, and often detached. The submucosa is firm, opaque, white or grayish, and glistening. Microscopically it is seen to be composed of white fibrous tissue arranged in irregular bands and networks. The fibres are rarely dense, but are usually well formed and developed, and only occasionally rudimentary and mixed up with many cells. Very little notice has been taken of the condition of the blood-vessels. In my own case they were comparatively few in number, and showed thickening of their outer coats and sheaths, but no signs of any endarteritis. No observer has succeeded in finding any structure distinctive of cancer. In a few cases, however, the embryonic nature of the growth has been suggestive of sarcoma; in my own case there was no resemblance whatever to either the one or the other. The muscular coat is thicker, firmer, and paler than normal; it is penetrated by fine bands of fibrous tissue, visible even to the naked eye, passing outwards from the submucosa to the subserosa. Under the microscope the muscular fibres are seen to be greatly increased in numbers, especially in the circular layer; and generally they show also an infiltration of fibrous tissue, either restricted to definite strands, or diffusely and intimately mixed up with the muscle fibres, as, for example, in an old myoma uteri. The serous coat with its subserosa is generally thickened, often considerably, so that its surface has a dull white opaque look instead of its normal clear and shining appearance.

Other signs of extensive or subacute peritonitis are not wanting in many cases; for thickenings and opacities of the gastro-hepatic and gastro-colic omenta, of the tissues behind the stomach, and other tissues around or of the peritoneum elsewhere, have been observed. Exudation of lymph over the intestinal coils, fibrous adhesions, ascites, general dropsy, or cirrhosis of the liver may coexist with it.

Ætiology.—The number of cases recorded is small, about two dozen

altogether; and the particulars given do not allow us to draw very trustworthy inferences from many of them. They are almost equally divided between the two sexes, and the average age of occurrence in man seems to be about 50, and in woman somewhat under 40. The most important point is the distinction of this lesion from cancer. All the earlier writers (Andral, Cruveilhier, Brinton, Habershon, Fox, Wilks) have drawn the distinction; though it must be admitted that the microscopic part of the evidence upon which they relied was imperfect. They considered the disease as a primary cirrhosis of the submucosa of a simple inflammatory nature, homologous with the common cirrhosis of the liver; and regarded the changes seen in the other coats as subsequent and subsidiary thereto. Recently Bret and Paviot have sought to prove that the lesion is really cancerous; they found their contention, not so much upon the microscopic appearances of the submucosa, which they admit even in their own cases did not show a structure clearly indicative of cancer, as upon the condition of the perigastric lymphatic glands — which did show characteristic epithelial cells — and upon the presence of metastatic growths. They argue, further, that all previously reported cases, from their strong similarity to their own, were really of the same nature. This is a doubtful inference. They have succeeded in showing only, what was long believed, that a cirrhotic atrophic cancerous infiltration of the walls of the stomach may so simulate a simple cirrhosis of that organ as to be indistinguishable from it, save by a most careful and searching microscopic examination. It is not improbable that several of the cases recorded as simple may have been cancerous; but it by no means follows that all of them were so. All the previous observers cannot have fallen into this error. In my own case the wide extent of the lesion made cancer seem improbable, and microscopic examination showed beyond all doubt that this impression was correct. Nevertheless, while awaiting further evidence, we may remark in passing that there is nothing inconsistent or improbable in the supposition of a simple cirrhosis. We meet with a like condition in other organs, as, for example, in the liver and kidney; and in the deeper layers of the skin beneath the epithelium we find dense fibrous growths, either localised or diffuse, in keloid and scleroderma respectively, for the origin of which we are unable to account. Alcohol was regarded by Brinton as a likely exciting cause, but little dependence can be placed upon it, for not only have we the fact that the disease is rare as compared with the frequency of over-indulgence in alcohol, but we find that in the great majority of cases there is no history of alcoholism. Syphilis is shut out in a similar manner. An attempt has been made to show that the disease spreads from without, and chronic perigastric peritonitis is pointed to as the prior condition, and as the cause of the stomach changes. It is, however, quite as probable that the changes are concomitant, or even that in some cases the sequence is reversed. There is another and quite distinct form of cirrhosis which frequently follows chronic gastric catarrh and atrophy of the glands. Here the first change is in the mucous coat, which

becomes thinned, atrophied, and sometimes sclerosed. The inflammation extends to the submucosa, generally in an irregular way, giving rise to patches of fibrous thickening, sometimes of considerable size, which are most frequent and abundant in the region of the pylorus, and may lead to a certain amount of hypertrophy of the muscular coat. The cavity of the stomach is generally dilated. In a well-marked case under the care of Prof. Clifford Allbutt the process seemed to have taken its rise in the scar of healed ulcers, whence it invaded the whole stomach. The organ was much contracted, and the patient ultimately died of pyloric stenosis. The ulcers—simple ulcers which healed soundly—occurred between the ages of twenty and thirty, and death ensued from stenosis some ten years later. Judging from the scars the ulcers had been of considerable extent and severity, and the result a sort of keloid.

Symptoms.—It would appear that in some cases the disease has run its course without giving rise to any symptoms, death being due to some intercurrent malady. In such cases the changes are said to be slight. In the great majority of the cases well-marked symptoms have come on, sometimes suddenly, sometimes insidiously. Pain in the epigastrium is the chief of these. It varies greatly in different cases, and at different times in the same case. It may be slight or very severe. It comes on at first only after food, generally immediately after, and usually becomes more severe as the case progresses. Vomiting is also very frequently present; it sets in after the pain, at first occasionally—say, three times a week or so—but gradually increasing in frequency and severity, until it may occur many times a day. The vomited matters may consist of the ingested food, of clear watery mucus, or later in the disease also of coffee-ground material, or even pure blood. The hæmatemesis, which is due to the congestion and sometimes to the ulceration of the mucous coat, is occasionally copious. Sometimes there is tenderness on pressure over the epigastric region; but this is often a delayed symptom. The appetite is bad, and there is usually constipation, at any rate for a considerable time. There is at the same time a steadily advancing anæmia and emaciation. Since these are symptoms equally characteristic of chronic gastric catarrh and of perforating ulcer, it is obviously impossible by their means to diagnose between them; but as the gastric cirrhosis advances a tumour becomes palpable in the epigastric region; occasionally below the ribs on the left side, and exceptionally in the left side: it varies in size, is round or sausage-shaped, firm and smooth, is fairly movable from side to side, and also slightly from above downwards. If caught with the hand and prevented from moving when deep respirations are taken, it is said to slip immediately upwards when liberated. Percussion over the tumour is never dull, and generally gives a resonant or moderately resonant note. An ascites has been found in some cases, and a general dropsy in others. Cirrhosis of the liver is occasionally present also. The anæmia and emaciation advance steadily, and the vital powers gradually diminish; the patient usually dies from asthenia. The rapidity of the process seems to vary greatly; it generally lasts for

many years, though in some cases the symptoms date back for only a few months, or even less. It is probable that the disease is then fairly well established before it gives rise to any symptoms. In Dr. Allbutt's case the symptoms were those here described, but no dropsy appeared. Not only could the stomach be manipulated and its size ascertained, but its movements were very active, and conspicuous in both directions.

Diagnosis.—The appearance of the tumour is the first trustworthy indication of the nature of the disease; it enables us to set aside simple gastric catarrh and ulcer. The presence of a tumour is equally characteristic of cancer; but from this disease we may be able to distinguish it by the smoothness, mobility, and semi-tympanitic note of the tumour, the time of onset of the vomiting and the pain, the infrequency of hæmatemesis, the absence of signs of metastasis, and the long duration of the malady. Peristalsis may be visible. We have as yet no information whether free hydrochloric acid is present in the gastric juice or not. Attention to this point might prove of value. Further, the stomach may be washed out and an attempt made to ascertain its capacity; if it does not hold more than a few ounces the diagnosis will be supported.

Treatment.—Under the ordinary treatment of gastric catarrh or of ulcer the earlier symptoms often improve greatly for a time. Later, when the nature of the disease becomes probable, the chief indication is to support the patient's strength. A mild and bland nutritious diet should be given in small quantities, and repeated at more frequent intervals than usual. Milk, combined at times with cream, strong soups, beef essences, raw minced beef, and so forth, should be chiefly relied upon. Nutrient enemas may also be used. Repeated washing out of the stomach with dilute soda solution has been tried in some cases with a favourable result. The quantity of fluid introduced should, it is said, be increased gradually until the patient can bear about one litre with comfort. Among medicines opium is the most highly recommended. Cocaine internally, leeches, or mustard blisters, or hot applications to the epigastrium, may also be tried for the purpose of alleviating the pain. Gastric tonics may do some good. Cases of recovery are reported by Schnetter and also by J. W. F. Smith; but in estimating their value the difficulties in the way of an indubitable diagnosis during life have to be considered.

REFERENCES

1. ANDRAL. *Précis d'anatom. path.* t. i. p. 47.—2. BRET and PAVIOT. *Revue de médecine*, May 10, 1894, p. 384.—3. BRINTON. *Diseases of the Stomach*, 1864.—4. CRUVEILHIER. *Anat. path.* 1856, t. iii. p. 599.—5. FOX, WILSON. *Diseases of the Stomach*, 1872.—6. HABERSHON. *Diseases of the Stomach*, 1869.—7. HANOT and GOMBAULT. *Archiv. de physiol. normale et path.* 1882, vol. ix. p. 412.—8. JONES, HANDFIELD. *Pathological and Clinical Observations respecting Morbid Conditions of the Stomach*, 1854.—9. KAHLDEN. *Centralblatt f. klin. Med.* 1887, No. 16.—10. SCHNETTER. *Deutsches Archiv f. klin. Med.* Bd. xxxiv. S. 632.—11. SMITH, J. W. F. *Edin. Med. Journ.* 1872, vol. xviii. p. 521.—12. SNELLEN. *La Lancette Néerlandaise*, 1855, and *Canstatt Jahrber.* 1856.—13. WILKS. *Trans. Path. Society London*, 1861-62, vol. xiii. p. 83.

R. F. C. LEITH.

SEA SICKNESS[1]

SYNONYMS.—Fr. *Mal de mer;* Germ. *Seekrankheit;* Lat. *Nausea;* Gk. *Nausia*, or *Nautia.*

Definition. — A functional disorder of the stomach often associated with giddiness, which affects travellers by sea, animals as well as man, and is chiefly felt by persons unaccustomed to its movements.

Etiology.—*Immediate causes.*—The primary cause of sea-sickness is the irregular, unremitting, undulating motion of the vessel; or, in language more precise, the oscillation that occurs about its longitudinal and transverse axes. Of these two movements the pitching on the transverse axis is more unpleasant than the rolling on the longitudinal; but the motion is still worse when the two movements are combined in the tumult called a "wobble": the principal uneasiness is felt at the moment of descent. The amount of suffering varies in direct proportion to the degree, the frequency and the suddenness or quickness of the motion. Generally speaking, it is greater in a sailing boat than in one propelled with oars, and in a screw-propeller than a side-wheel steamer; it often happens that one who is accustomed to the movement of a steamer, sailing ship, or row-boat becomes sea-sick when transferred from one into the other, the character of the motion being very different.

Much depends upon the weather and consequently on the season of the year; upon the situation one may occupy on board, the amplitude of motion being greater the farther one removes from the centre of the vessel; the direction of the long diameter of the bunk or berth, which should be always placed fore-and-aft and not athwartship; and upon the state of the atmosphere below: when the air is stuffy, close, and saturated with the smell of cooking, of paint or driers, of heated oil, of bilge-water, of vomit or other excrement, it is almost insupportable; and those who are inclined to be sea-sick much prefer to remain on deck. In ships which are or have been used as cattle-ships the odour of the manure, or of the disinfectants used in order to conceal it, is often very disagreeable. These nuisances however, offensive as they are, together with the disagreeable sounds of vomiting and retching, the thud, the occasional racing and the vibration of the engines, the sight of swinging, swaying, oscillating objects, and the difficulty or impossibility of obtaining any rest even in the horizontal posture, can only be regarded as secondary or subsidiary causes of sea-sickness.

Remote causes.—The prime of life is the period most obnoxious to sea-sickness; infancy, childhood, and declining years are comparatively

[1] Although sea-sickness is no longer in the index of the nomenclature of diseases, it seems to me to have so much practical importance as to entitle it to specific consideration. —ED.

free. Women being less active, less accustomed to vicissitude, and less inured to travel, generally suffer more than men; at the climacteric period this proclivity is increased. The bilious temperament is more conducive to it than the phlegmatic, and the nervous than the sanguine; in respect of race and nationality the people of the northern parts of Europe suffer less than those belonging to the southern, the Celtic and the Scandinavian races less than the Teutonic, and the Teutonic than the Latin; the Americans are said to suffer more than we do. Townsfolk generally suffer more than country people; but an exception must be made in favour of those who dwell upon the sea-board, and those who take a pleasure in aquatic exercises. People who pursue an active outdoor life suffer less than those who work indoors; keen and eager sportsmen suffer very little. Those of spare and frugal habits undoubtedly fare better than those addicted to the pleasures of the table; nothing favours an attack more than a debauch. Deaf-mutes and the insane are said to be exempt from sea-sickness; but so are many others who possess the soundest health of mind and body, and have no impairment of their senses. Imagination often plays an active part in its production.

Symptoms.—The pleasurable sense of physical and mental exhilaration which is the first effect produced by a moderate degree of motion on the water soon gives way to a void or empty feeling at the stomach and a craving like that of hunger, but with no desire to partake of food. A feeling of oppression, weight, or sinking at the epigastrium; an increase of the mucus and saliva in the mouth and throat which, instead of being swallowed by degrees, is gulped down; an occasional gape or yawn, sometimes followed or preceded by a sigh; a sudden pallor of the face; a little choking cough or hiccough; a hawking up and expectoration of viscid phlegm or mucus; a sense of squeamishness, as though any moment vomiting might occur; flatulent or sour eructations; together with the general state of mind and body consequent thereon, namely, physical weakness, mental and nervous depression, giddiness and faintness, are sure and certain signs of a disordered stomach, and constitute the premonitory symptoms or prodroma of an attack.

The principal symptoms, when the attack has once set in, may be divided for the purpose of examination into two groups: those belonging to the stomach and those belonging to the head. The first or gastric group comprises nausea, vomiting and retching, total loss of appetite, and constipation; the second or cephalic group, which the Germans call the "psychic," consists of faintness, giddiness, and headache. In some persons the gastric group predominates, in others the cephalic; but in the majority of cases both the stomach and the head participate alike, and comparatively few persons suffer from the one group to the exclusion of the other. I shall have occasion presently to make further reference to this arbitrary distinction between the two, but first I must describe the symptoms individually.

1. *Nausea.*—This was the name given by the ancients to denote sea-[illegible] is now used only to denote its leading symptom. Common

enough in febrile complaints, in severe inflammatory diseases, in organic disease, in sudden and violent shocks to the system generally, and in divers forms of reflex irritation and excitement, this distressing feeling frequently constitutes the principal and sometimes the only symptom of sea-sickness. In susceptible people it may be provoked by the sight or even by the mere idea of undulating movements.

2. *Vomiting and Retching.*—Sea-sickness may begin and end in nausea, and nausea alone; but sometimes, and especially in the case of children, it consists of vomiting only, which may occur quite suddenly and unexpectedly, without any previous warning or indication, and without causing any inconvenience. But as a rule the vomiting is associated with nausea, headache, heaviness of the head, and giddiness, and is followed by most violent and distressing retching; the little nourishment that can be taken is immediately thrown up, but subsequently only the mucus and saliva that is swallowed, along with gastric fluid and green or yellow bile; later, pure bile comes up alone. Meanwhile the patient's strength begins to fail, he falls into a low despondent state, becomes indifferent alike to circumstance and fate, and feels inclined to wish (if wish he can) that death itself might end his sufferings.

3. *Loss of appetite and constipation.*—States of pleasure, we are told, are concomitant with an increase, and states of pain with an abatement of some or all of the vital powers; the same is true of comfort and distress. This will account in great measure if not entirely for the anorexia. Symptomatic, like the nausea, of many other forms of gastric disorder, both functional and organic, in sea-sickness nausea and anorexia attain a quite exceptional degree and out of all proportion to the actual gravity of the case. Not only does the appetite for food continue in abeyance as long as the disorder lasts, but so also does that for every other kind of enjoyment or indulgence; even sleep, which often cannot be obtained until the vital powers are exhausted, is frequently disturbed and fails to renovate the failing powers of the system. In persons of a delicate, weak, and nervous constitution, who have but slender stock of health and strength wherewith they may encounter such attacks, the recovery is often tedious and the effects are sometimes serious. Constipation, though partly due to lack of proper stimulation of the bowel by the gastro-biliary secretions, may partly be accounted for by the uneasy and distressing motion. In railway travelling the constant oscillation, vibration, and concussion to which the body is exposed, while most destructive to the plant, are more or less injurious to the animal system; not only are they most fatiguing to the body and trying to the eyesight, but they delay the process of digestion itself, and, by paralysing the natural peristaltic action of the gut, constipate the bowel and sometimes end by causing sickness. It is said that these and other ill effects of railway travelling have been considerably diminished upon certain lines by the introduction of the bogie car. So too in sea-sickness the motion causes constipation, and the constipation serves to augment the other symptoms and also in a measure to provoke them. The amount of urine also is diminished,

but this depends more directly on the vomiting and the small amount of fluid that is swallowed.

4. *Giddiness and Headache.*—The headache is situated generally in the frontal region, though sometimes also in the vertical or occipital; it is either of a dull and heavy, or of an aching, throbbing character, and is usually accompanied by a feeling of fatigue or soreness, and by aching or neuralgic pains at the back of the orbit. In the paroxysms of retching and of vomiting, the eyeballs are protruded and feel as though they were starting from their sockets, and the vessels of the head and neck are swollen. Each of these conditions may and often does occur in any ordinary attack of vomiting, and it would be needless to describe them here were it not that on account of their pertinacity and severity they become more distressing and pronounced; in part they may be due to eye-strain, but they are mainly due to the disorder of the stomach. The giddiness or vertigo, however, which is not to be regarded as an integral part of the complaint, but only as accessory, may possibly be due to visual or perhaps to labyrinthine perturbations.

Which of these two affections, the cerebral or the gastric, is primary, or whether they are independent of each other, is still a matter of dispute. Probably there is a certain amount of truth in both of these conjectures, but few who contemplate the subject from a practical rather than a theoretical or even an experimental point of view can doubt that, even if they are related to each other, the probability is very much in favour of the gastric rather than the cerebral affection being primary. Giddiness, indeed, is often due to overloaded or disordered stomach, to gastric or to intestinal irritation; but in sea-sickness it is the symptom most readily relieved by lying down, and I therefore think it much more likely to be due, as Wollaston believed, to gravitation of the blood, combined of course with the sight of shifting images and disturbance of the equilibrium.

5. *General or constitutional condition.*—This has partly been explained in the statements made above. There are signs of shock or collapse of the nervous system, together with a quasi-febrile state; alternate heats and chills, with frequent chattering of the teeth and shivering, pervade the system; the eyes are dull and sometimes bloodshot from excessive straining; the countenance becomes extremely pale, inexpressive, and dejected; the complexion often of a greenish hue. The heart's action is depressed; the pulse and respiration quick and feeble; the skin cold and often clammy, while the tongue is moist but slightly coated. There is always some degree of hebetude; the mind is utterly indifferent to all around, no consecutive train of thought can be pursued, and the functions of the brain appear to be almost suspended. There is a general feeling of soreness all over the body, a sense of weariness and fatigue, and always more or less exhaustion and prostration.

Course and Progress.—If the voyage be a short one the symptoms usually subside as soon as it is over; nausea, giddiness, and headache may, and after a first voyage in particular often do, continue for a day or two

and sometimes even longer; but they are none the less on that account amenable to treatment. People sometimes have recourse repeatedly to these short voyages or pleasure trips upon the coast in order to recover health; the rougher the weather and the sicker they become, the better are they pleased. Possibly in cases where the liver is inactive and the digestive power feeble, or where the one is engorged and the other overwrought, such voyaging may be beneficial; and it was probably on this account that Burton recommended it. In longer voyages four successive stages may be recognised, namely, those of depression, exhaustion, reaction, and convalescence; but the symptoms altogether rarely last for longer than a week, very often they remit in the course of three or four days, but recur if the weather become worse or the bowels costive. The worst time is in the morning, the patient almost always feels better towards the evening; but the reaction is gradual and progressive, and, with careful regulation of the diet and attention to the bowels, the relief experienced on getting accustomed to the motion of the vessel is permanent. In some few cases the symptoms never cease until the end of the voyage, even though it be a long and tedious one like that to India, China, or Australia; and we sometimes meet with persons, generally women, who having come on board in feeble health or with a delicate constitution never leave their beds until they quit the vessel: on the other hand, such persons often find their health very much improved, if not entirely restored. In women, too, the menstrual molimina are usually more severe, the flow may either be augmented or diminished or sometimes interrupted altogether, and the period is frequently most irregular. In pregnant women, contrary to what might naturally be expected, miscarriage is not more liable to occur than it is ashore; but extra care should be always taken of them so as to avoid the risk of injury, or, if labour should occur, of post-partum hæmorrhage. It has been stated by Dr Barnes that the use of a suitable pessary will often completely relieve an attack of sea-sickness in women who have some displacement of the womb.

Complications.—Continued cases, such as those above alluded to, and others of a less protracted character, not infrequently assume a *low or adynamic form* and require a considerable amount of care. The vomiting may abate somewhat, but nothing will remain upon the stomach, and ultimately the patient is quite unable to swallow any solid food and is averse from taking any liquid nourishment; he will only suck a little ice. The nausea is constant, the throat and mouth are parched and dry and the skin likewise, the temperature is above the normal, the pulse quick, small and feeble, the respiration shallow and increased in frequency, the complexion sallow and the conjunctivæ tinged with bile, the countenance expressive of anxiety, and a mild delirium, a very rare concomitant of sea-sickness, may supervene; in these cases there is always pain and tenderness of the abdomen, generally in the region of the stomach. These symptoms closely correspond with those of acute or subacute gastritis; and there is every reason to believe that in the act of vomiting

the stomach may be wrenched or strained, or the mucous membrane irritated by the action of the gastric juice.

Sea-sickness sometimes takes the form of *diarrhœa*.

Jaundice is very rarely seen except in cases where the liver has been previously deranged.

It is not at all uncommon for *delirium tremens* to occur at sea, though whether this be promoted by sea-sickness may be considered doubtful; it is in my opinion much more likely to depend on constipation of the bowels. *Insanity*, particularly monomania of a suicidal character, is not infrequent, though it does not always follow that it depends immediately upon the motion any more than the *delirium tremens* does; or that it was occasioned by sea-sickness. Women who may or may not be hysterical are sometimes seized with fainting fits or fall into a state of frenzy in consequence of the violence and continuance of the sickness, or of the extreme weakness and prostration resulting from it. All such patients should be watched most carefully, and if necessary locked up.

Without denying altogether the possibility of *death* from sea-sickness pure and simple, we must hesitate before admitting it as a fact. I believe that it may occur in consequence of the gastritis already mentioned, in consequence of syncope, or perhaps from sheer exhaustion. In cases such as these, if death occurred it might properly be attributed to sea-sickness as the primary cause; but it would perforce be necessary to quote the secondary and more immediate cause also. This would apply still more particularly to cases in which death occurs in consequence of the entrance of a piece of undigested food into the glottis during an attack of vomiting—the immediate cause of death being of course not sea-sickness but suffocation. In all such cases it would be advisable to make a necropsy, and to look for some sudden cause of death such as apoplexy, aneurysm, a large dilated or a fatty heart, aortic or mitral insufficiency, contracted mitral, pneumonia, ulcer of the stomach or bowel, a strangulated hernia or granular disease of kidney; but under any circumstances it would still be difficult to regard a disorder, due to nothing more than a perverted function, as a principal or essential cause of death.

Pathology.—Pathologically sea-sickness has been attributed to a variety of causes: it is not my purpose to discuss them all, but the following list will show how materially they differ in character from each other:—

i. Concussion of the brain (Wollaston, Alderson, Neuhaus); ii. Anæmia of the brain; iii. Hyperæmia or congestion of the brain and spinal cord (Chapman); iv. Functional impairment of innervation (Beard); v. Visual disturbances (Mayo, Wilson-Fox, Graily Hewitt, and others); vi. Labyrinthine disturbances (Irwin, James of Harvard, and others); vii. Molecular concussion of the viscera (Rosenbach); viii. Concussion of the sympathetic ganglia or plexus—solar, semilunar, coronary, or splanchnic; ix. Disturbance or succussion of contents of stomach (Glynn Whittle); x. Disturbance of equilibrium (Mayo and others); xi. Regurgitation

of bile into the stomach; xii. Disagreeable odour from the bilges (Rawlinson).

Assuming for the sake of argument that any one or all of these conditions may be present during an attack, they may severally be put aside as insufficient to account for a disorder which betrays so many partialities, such obvious anomalies, as this; or to explain the reason why so many persons are exempt, or how it is that practically all recover. Moreover we should surely find now and then that some ulterior effect, some morbid consequence, some organic lesion would ensue, particularly in cases where any local or constitutional proclivity to disease exists; in my own experience this has not happened, and if it did happen we should probably hear much more about it. As to the "visual disturbance" hypothesis the late Dr. Graily Hewitt, who was one of its warmest advocates and made a series of experiments with the object of determining the influence exerted by the sight of shifting images, came to the conclusion that although they added much to its discomfort they could not be held entirely responsible for producing the malady, and he admitted that the blind are not exempt. Driven from this untenable position, those who have been looking to the nerves of special sensation to supply them with a clue have resorted to the ear—a clue, in my opinion, equally deceptive—and pursue a similar train of reasoning to that pursued by Dr. Beard, who argued that, since vomiting is one of the symptoms of concussion of the brain, it must in sea-sickness be due to a series of mild concussions of the brain. These authors, however, deal mainly with the vertigo and assert that, as vertigo is a prominent condition of sea-sickness and is often due to some affection of the semicircular canals, the secret of sea-sickness is to be found in this locality; they attribute it either to some mechanical disturbance of the endolymph or to an irritative hyperæmia of the membrane, and recommend for its abatement the use of counter-irritants behind the ear. In support of this hypothesis Dr. James of Harvard states that those who suffer from deaf-mutism are comparatively if not absolutely free from the complaint, and that they also suffer less when seated in untwisting swings. But even if it could be proved that the vertiginous effects are really due to these disturbances it would be quite unwarrantable to conclude that sea-sickness itself is due to them. The truth appears to be that both the visual disturbances and the labyrinthine (if such there be) unite with the other uneasy, disagreeable sensations that occur on board ship in making matters worse, in adding to the discomfort of the sick, and in frequently precipitating an attack, but that they cannot be regarded as prime or essential factors in producing it.

Let us briefly refer to what Wollaston said upon the subject. He had observed that the mercury rose higher in the tube of a barometer when the ship began to fall; he concluded that the action of the blood upon the brain would be identical, and that this would cause a pressure or a blow upon the brain which by frequent repetition would result in vomiting and nausea. He experienced a necessity in his own case, and

believed that others would perceive the same, to draw a deep breath at the moment of descent; this he attributed to an instinctive effort so to increase the capacity of his chest that the blood might flow more freely into it and thus relieve the pressure it would otherwise exert upon the brain; he found that the method gave him great relief, and he thought that this would prove to be the proper explanation and provide the proper remedy for sea-sickness. Alderson, who wrote in 1872, also expressed his firm conviction that this was the true explanation, and he argued strongly in its favour. But if it be correct the remedy would be obvious enough; the horizontal posture with the head lower, or, as Neuhaus puts it, to lie upon the side with the head bent upon the chest and the knees drawn up towards the chin, should be sufficient to arrest or to prevent it. The horizontal posture does relieve it certainly, but not so much nor so effectually as it should do if the explanation were correct; it may be partly true but it cannot be sufficient. Wollaston himself admits that he first perceived the inclination when waking up from sleep, but it evidently did not occur to him that the very fact of the occurrence or continuance of sea-sickness when in this position is a strong if not insuperable argument to the contrary. Had Wollaston been acquainted with the researches subsequently made by Marshall Hall and others into the phenomena of reflex action, or carried his own investigations a little further, he would probably have found some cause to modify his views. That Hall himself considered sea-sickness to be a reflex act is certain, for he particularly refers to it as being so.

Deep inspiration taken at the moment of descent and constantly repeated will indeed provide the proper remedy, but principally, I apprehend, because it thwarts the tendency to the sudden and impromptu closure of the glottis which instinctively occurs in sea-sick persons. This spasmodic action of the glottis, either by direct or reflex action, at once excites the various conditions and sensations that immediately precede and accompany the vomiting, the choking being probably its direct precursor; and by constant repetition it soon induces the usual train of symptoms.

Closure of the glottis (or larynx), as a voluntary or spontaneous action, serves principally as an aid in making or increasing certain efforts; but whenever danger or discomfort threaten suddenly or alarmingly, as is commonly the case at the moment of descent, it occurs unconsciously, instinctively, and with great rapidity; and I believe that its constant repetition is the cause of all the trouble. It may be overcome by drugs, by habit, or by strong determination; or may happily be avoided altogether, as in the case of people who possess immunity. The difference, then, between a good sailor and a bad one appears to me to lie mainly in the difference in their mode of respiration; and the cause of the complaint seems to be seated neither in the stomach nor in the brain, but in the throat.

Considering the very delicate sensibility of the larynx and the

pharynx, their close proximity, their direct communication with each other, their connected innervation, and the immediate contiguity if not the actual identity of the cerebral and probably also of the spinal centres for respiration and for vomiting, we can readily understand how either momentary or constant irritation, annoyance, and distress of one may either by direct or reflex action be transmitted and produce effects upon the other.

Treatment.—1. *Prophylactic.*—This consists mainly in careful regulation of the bowels and adjustment of the diet for a day or two at least, if possible for a week, before the beginning of the voyage. The diet should be of the lightest possible description consistent with the maintenance of health, and those who desire to avoid sea-sickness altogether cannot do better than adopt a purely vegetarian diet for the nonce; all alcoholic drinks should be forbidden, and nothing should be used either as a condiment, food, or drink which has the slightest tendency to confine or constipate the bowels; an antibilious pill or two should be taken if necessary every other night so as to bring the stomach and the liver into thorough working order. If the voyage has to be begun without much time for preparation, then one blue and two compound rhubarb pills should be taken the night but one before embarking; or, if greater expedition be required, a dose of Hunyadi or of Friedrichshall water, or some gentle saline aperient, such as the Carlsbad or the Glauber salt, should be taken warm before breakfast. The traveller, however, should never go on board upon an empty stomach; the last meal should be a hearty one and a glass of stout may be allowed. It has been asserted that the steady use of bromides for several days before the voyage will render people less susceptible to sea-sickness, and this I think may be admitted; on the other hand, the indiscriminate or continuous use of them must be condemned. The late Dr. Hammond of New York expostulated very strongly against this practice, and his experience is fully confirmed by many other medical men both ashore and afloat. The bromides all exert an anæsthetic action on the larynx and the pharynx, and are often usefully employed in examinations and in operations about these parts, and also in pertussis; they were originally recommended for the use of travellers with the object of diminishing the excitability of the nervous system generally, but their chief utility, in my opinion, is due rather to their specific action on the throat, and, if my view of the pathology be correct, they would probably be quite as serviceable and at the same time less injurious were they in future to be used in the form of lozenges or as gargles. Atropine and hyoscyamine, which also act in some degree upon the throat, have been used with good effect; they may be taken in the form of sulphate once or twice a day, the dose of the atropia salt being from $\frac{1}{150}$ to $\frac{1}{100}$ of a grain and that of the hyoscyamine salt from $\frac{1}{100}$ to $\frac{1}{50}$ of a grain. When the vomiting has set in the hypodermic method may be used with more success. This applies to cocaine also, and to the use of ergotine for menorrhagia.

Chloral and chloralamide are extensively employed as anodynes or hypnotics; and "chlorobrom" (a mixture of chloralamide and a bromide) is often most successful in staving off sickness during a short voyage of from six to twelve hours. Chance found that a dose of laudanum taken before he went on board made him absolutely indifferent not only to the motion of the vessel but also to the recollection and the dread of its effects, and he used it subsequently in smaller quantities with equal benefit. Whatever drug is used should always be prescribed with moderation. A dose or two of a sedative taken to surmount the discomfort of a short passage may be proper enough, while it would be improper to use such means continuously or frequently on a long voyage. If sea-sickness cannot be prevented without much lowering of the system, and of the nervous system in particular, it will be more expedient to refrain altogether from any interference and to leave the case entirely in the hands of Nature. The use of a tight abdominal bandage is said to be of service on account of the restraint it puts upon the movements of the solid viscera; it would certainly prove less irksome if applied a day or two beforehand and tightened up on embarking.

It might be worth while to try the effect of hypnotic suggestion.

2. *Remedial.*—To lie flat upon the back with the head upon the level is undoubtedly the posture most approved of by the public; it should be assumed before the vessel leaves her berth or moorings. Long preferred to let the head hang down a little; this position of extension is the one that Howard recommended in cases of suspended animation, and, involving as it does a wider opening of the glottis, it would, I think, be more effectual. Mackenna thought it best to lie upon the [right] side and thus to keep the head below the axis of the body; the curvilinear attitude, recommended by Neuhaus shortly afterwards, has already been referred to. Chapman's ice-bag to the spine, though once a fashionable remedy, was condemned by Fordyce Barker and has fallen into desuetude, and the use of ice is now confined to sucking it or to sipping ice-cold water or champagne in order to relieve the dryness of the mouth and throat and to quench the thirst. Considerable relief may also be obtained by sucking lemons or, when these are all consumed, by using lime juice, which when mixed with soda-water makes a pleasant drink.* Beef tea, beef or calf-foot jelly, veal or chicken broth and a little gruel, arrowroot, or barley water often constitute the only nourishment that can be taken.

Still it is always best to bite or at least to suck a crust of bread, a piece of toast, a biscuit or a rusk; while a sip or two of port, champagne or even stout is often invaluable.

During a slight attack, or when the patient is recovering, condiments such as curry, Worcester sauce, and Cayenne pepper, though injurious in the worst stage, become very useful; while gin-cocktail or l'élixir végétal de Grande Chartreuse may be sometimes used with great advantage. To take a draught of cold sea-water, to bathe the hands and

face in it, and to face the air on deck as much as possible are simple measures of the greatest service.

Unless, then, it be to get over short voyages, the less we resort to drugs the better; they increase the nausea and oftentimes the vomiting, which is both discouraging to the patient and disappointing to the doctor; the warmest advocates of specific remedies candidly confess that they are of comparatively little use unless the treatment is begun before the voyage, a course to which very few who are either bent on business or on pleasure are willing to submit. Those who can resist the inclination to lie down and who keep continually moving, endeavouring to adapt themselves and especially the mechanism of their respiration to the motion of the vessel—which can best be done by means of calisthenics in the open air, by shouting or by singing—will soon become accustomed to it. If we use a drop or two of chloroform, a few grains of chloral, a little bismuth or magnesia, ingluvin or pepsin, or some peppermint or ginger; employ an occasional cathartic or some aperient saline and secure a daily movement of the bowels, if necessary by the use also of an enema or suppository, we shall probably succeed in the majority of cases; others must be treated after their special features; but of no bodily disorder can it be more truly said—"Our remedies oft in ourselves do lie."

J. R. STOCKER.

REFERENCES

1. ALDERSON. *Observations on Sea-Sickness*, Lond. 1872; *Brit. Med. Journ.* vol. i. 1872.—2. BARKER. *On Sea-Sickness*, New York, 1870; *New York Med. Journ.* Nov. 1868; Ranking's *Abstract*, vol. i. 1870.—3. BARNES. *Brit. Med. Journ.* vol. i. 1892.—4. BEARD. *Practical Treatise on Sea-Sickness*, New York, 1880.—5. BELL. *On the Hand*, 8th ed. Lond. 1877, pp. 148, 156.—6. BRUNTON. Quain's *Dictionary of Medicine*, 2nd ed. Lond. 1894, art. "Vomiting."—7. CARPENTER. *Principles of Human Physiology*, 6th ed. Lond. 1864, §§ 73, 495, 521, 526.—8. CHANCE. *Brit. Med. Journ.* vol. ii. 1881.—9. CHAPMAN. *Sea-Sickness*, 2nd ed. Lond. 1868; *Med. Times and Gazette*, vol. ii. 1864; Ranking's *Abstract*, vol. i. 1869.—10. DRUITT. *Surgeon's Vade Mecum*, Lond. 1865, c. xv. § 3.—11. DUTTON. *Sea-Sickness.* Plymouth, 1890.—12. FOSTER. *Text-Book of Physiology*, 4th ed. Lond. 1883, bk. iii. c. vi. § 2.—13. FOX. *Diagnosis and Treatment of Dyspepsia*, Lond. 1867, p. 63.—14. GIRALDÈS. *Journ. de Thérapie*, Nov. 21st, 1874.—15. HALL. *Diseases and Derangements of the Nervous System*, Lond. 1841, p. 104.—16. HAMMOND. *New York Med. Record*, July 2nd, 1881.—17. HELMHOLTZ. *Optique physiologique*, MM. Javal et Klein, Paris, 1867, p. 769.—18. HEWITT. *Brit. Med. Journ.* vols. i. and ii. 1892.—19. IRWIN. *Pathology of Sea-Sickness*, Lond. 1881; *Lancet*, vol. ii. 1881; *Influence of Sea-voyaging on the Genito-urinary functions*, New York, 1885.—20. JAMES. *Amer. Journ. of Otology*, vol. iv. 1882; *The Practitioner*, vol. ii. 1883; *Boston Med. and Surg. Journ.* vol. cxvi. 1887.—21. KIRKES. *Handbook of Physiology*, Lond. 1867, c. xvi.—22. LEISER. *Brit. Med. Journ.* vol. i. 1888.—23. LONG. *Ibid.* vol. ii. 1880.—24. LORTET. *Lyon Médical*, Sept. 1869; *Comptes Rendus*, vol. ii. 1869.—25. MACKENNA. *Brit. Med. Journ.* vol. ii. 1880.—26. MACPHERSON. *Brit. and For. Med-Chir. Rev.* vol. ii. 1876.—27. MARSHALL. *Outlines of Physiology*, Lond. 1867, pp. 252, 265, 341, 348, 520.—28. MAYO. *Outlines of Physiology*, 4th ed. Lond. 1837, c. x. § ix.; see also *Index of Original Observations*, Pref. p. 8.—29. NEUHAUS. *Berl. klin. Woch.* 1885.—30. RAWLINSON. *Report on Quarantine*, Lond. 1849.—31. ROSENBACH. *Berl. klin. Woch.* 1891.—32. ROSSE. Buck's *Reference Handbook of the Med. Sciences*, New York, 1888, vol. vi.—33. STOCKER. *Lancet*, vol. ii. 1881; *New York Med. Journ.* Feb. 1882; *Nautical Magazine*, Oct. 1891.—34. TURNER.

Buck's *Hygiene and Public Health*, Lond. 1879, vol. ii.—35. WHITLA. *Dictionary of Therapeutics.*—36. WHITTLE. *Brit. Med. Journ.* vol. ii. 1880.—37. WILKS. *Ibid.* vol. ii. 1875.—38. WILSON. *The Ocean as a Health Resort*, Lond. 1880.—39. WOLLASTON. *Philosophical Transactions*, 1810.—40. ZOUCHE. Quain's *Dictionary of Medicine*, Lond. 1894, art. "Sea-sickness."

J. R. S.

MOUNTAIN-SICKNESS

SYNONYMS.—Fr. *Mal des montagnes ;* Germ. *Bergkrankheit.*

Short Description.—Lassitude; quick and gasping respiration increased on the slightest exertion, and headache, arising in all men at an altitude of 16,500 feet at most.

In this description are contained all the symptoms which are properly designated by the name mountain-sickness; nausea and vomiting, which are popularly supposed to form a necessary part of the group, are incidental, and probably due to other causes.

Causation.—The causes to which mountain-sickness has been attributed are fatigue, indigestion, diminished atmospheric pressure, heart failure, and deprivation of oxygen. In the light of recent observations it seems probable that the immediate and sufficient cause is deprivation of oxygen. It is true that diminished barometrical pressure, fatigue, indigestion, and heart failure may contribute to produce a state resembling mountain-sickness, or loosely so-called, at a lower level; it is true also that at the higher level it is not always or generally easy to eliminate these factors in a given case: nevertheless, by careful comparison of records and of physiological experiments, these additional factors are proved to have subordinate or diverse values.

It would be difficult, and it is not of great importance, at any rate in this place, to define exactly the several causes of the confused effects of bad or imperfect training, of improper food, of casual indisposition, of oppression of the heart, and so forth, which may and often do appear in the course of mountain expeditions undertaken by persons in bad condition; and it is manifest that in these persons such disturbances will come on at an earlier stage than in those better fitted for the work: I repeat, however, that these phenomena are inconstant, that many of them have no necessary connection with mountains, and that, on the other hand, the symptoms given in the short description are constant, and are characteristic of all high mountain ascents.

I will now shortly consider each of these alleged causes separately. For the supposition, many years ago, of *heart failure* as a cause of mountain-sickness I am chiefly responsible. That dilatation of the heart does occur in mountain-climbing has not been disproved by subsequent observers, rather the contrary; my observations on this subject have

indeed been corroborated more or less by Conway, Roy, and others; but I now admit that this change, if often coincident with mountain-sickness, is not the direct cause of it. Sir Martin Conway's admirable sphygmograms, taken at my suggestion on the Karakoram-Himalayas, have settled this point against me; moreover, I had become distrustful of my own explanation on other grounds. As I shall return to this subject in an article for this work on "Overstrain of the Heart," I need not detain the reader further in respect of it at present.

Fatigue is from the nature of the case a very difficult factor to eliminate; fatigue products must almost always enter into the causation of particular cases of distress during high mountain excursions. Still, it did not avail Mr. Whymper that on one occasion he rode on horseback up to an elevation of 16,500 feet; mountain-sickness was not thereby averted. Again, mountain-sickness is mitigated instantly on remittance of effort, and aggravated as instantly by its resumption, perhaps even on so slight an effort as the adjustment of the screw of an instrument of observation; this, if not conclusive, strongly suggests some cause more immediate than the circulation of a poison in the blood. Again, fatigue products are formed and infect the blood in exercises other than mountain excursions, and have been so far studied by many observers that their effects are fairly well known, and can be distinguished from mountain-sickness in the strict sense in which I now use the name. Arctic explorers, great as is their labour, do not suffer from mountain-sickness proper, and, as I shall show, the conditions of aeronauts are not altogether comparable with those of the mountaineers.

Indigestion, again, has been regarded as a potent cause of the malady we are now considering. And it is clear, from the experience of almost every member of the Alpine Club, that to mountain-climbers a healthy digestion is of fundamental importance. On a morning when his digestion is awry, the best of climbers may find himself reduced to impotence, at no very high level, by exhaustion, nausea, and even vomiting; on the other hand, great care in diet, a rigorous use of spare and simple food, may be as effective in postponing or preventing such distress. I well remember having to retire from an ascent of Monte Rosa on account of the repeated vomiting and prostration of a companion, one of the best climbers who ever trod the Alps, I myself being wholly free from the least discomfort; he was dyspeptic, and I was not, and mountain-climbing disagrees sadly with men who are out of condition. Partly because of my medical training, partly because I am never disposed to eat much when on the mountains, I have never suffered from this pseudo-mountain-sickness; and never having ascended higher than Mont Blanc I was long disposed to disbelieve in any such malady: I regarded the state rather as a compound made up of fatigue, dyspepsia, and heart failure. The experience of climbers—such as Sir Martin Conway, Mr. Whymper, and others—at far higher altitudes is, however, conclusive that there is a definite disorder thus designated, one which not only presents uniform features of its own, but also appears with a

remarkable uniformity in all persons, whatever their individual condition, at a certain altitude—about 16,500 feet: it is needless to say that it, or a modified form of it, may likewise come at a lower level if favoured in the individual case by other adverse conditions.

The next alleged cause to be discussed is the important one of *diminished barometrical pressure.* That such diminution is a potent cause of bodily distress and disease is well known in the so-called "caisson disease." But the symptoms of this affection present many differences from that of mountain-sickness. The factor in common between them is the change from a higher to a lower barometrical pressure. Now it is first to be noted that in caisson work the labourer is soon accustomed to the one pressure or the other. Give him time, and he is easy either in the ordinary pressure before entering the caisson, or in the high pressure within the machine. The danger lies in too rapid a transference from the one to the other. In mountain work the transference is more gradual; and the symptoms differ accordingly in the two cases. If it be said that the more gradual rate of the change in the mountaineer modifies the symptoms, they are so modified as to have lost all resemblance. On the other hand, the hardened mountaineer never loses the true sickness by any habituation. My old friend T. S. Kennedy with Fischer, when shooting sheep in the Nepaul Himalayas, camped for some days at an average height of something more than 18,000 feet; and Kennedy told me that so long as they remained at and above this level, the disinclination for exertion and the rapidity of the breathing were never mitigated, save by night and repose. Mountain-sickness does not consist, then, in sudden transference from one pressure to another, but is a persistent disability.

One more cause remains to be discussed, namely, *deprivation of oxygen;* this explanation was accepted by Professor Roy in his study of Conway's experience, and is also adopted by Dr. Hepburn in his careful and lucid discussion of the subject. To me also it seems that this cause will probably suffice to explain all the essential phenomena of mountain-sickness.

This being so, the subject loses its claim to a place in this section of this work; but, as the popular notion of mountain-sickness, even among medical men, is associated with sickness of the stomach, it seems to me that the present case is one of the many in which convenience is to be preferred to system. Dr. Marcet has shown that fatigue under exertion is due as much to the exercise of the cerebral motor centres as to exercise of the corresponding muscles; and that the sense of fatigue, too readily attributed perhaps to the accumulation of fatigue products, is also due to the supply of oxygen being inadequate to meet the demands consequent upon effort. Mr. Glaisher's experience in a balloon, and the results of rebreathing expired air, support the belief that an imperfect supply of oxygen to the brain molecules, although it does not prevent the formation of a volition, yet takes away the power to carry the volition into effect. It is a matter of common experience with us all that an effort is more

easily performed if a few long inspirations are previously taken; in this way the capacity for the effort can be increased. At 16,000 feet the oxygen per unit volume of the respired air has fallen to a definite point, to a degree apparently inconsistent with much activity, or even with strong volition. Moreover, there is a greater relative absorption of oxygen at pressures lower than 300 mm. of atmosphere; and, conversely, on surpassing 300 mm. the dissociation of oxygen is disproportionately increased. Roy, however, on consideration of the records, thinks that with care a farther height than Conway's 23,500 feet may be attainable. On the top of Pioneer Peak Zurbriggen exclaimed, indeed, that he could not have cut another step, but further step-cutting might not have been needed. In man, when the oxygen of inspired air is experimentally reduced to 10 per cent, symptoms of dyspnœa appear; and, as Hepburn says, at this external percentage the oxygen in the alveoli must stand at less than 10 per cent. Now this fall of oxygen corresponds to 3000 mm. total atmospheric pressure, and to a height of 17,000 feet. By habit the limits of toleration may be extended somewhat; but perhaps the toleration rather than the capacity would be increased—the distress would be less perceived, yet at the same time accurate observations might prove the functions to be not much less abnormal. In time, of course, the chest might enlarge in capacity as it does in the inhabitants of highlands; but Conway says that his coolies, who came from a region 10,000 feet in height, were affected, at 17,000 feet and upwards, much as the rest of his party. That diminished pressure in itself, apart from the oxygen percentage, has any share in the production of mountain-sickness is an assertion often made, but at present without the support of definite evidence. Hepburn argues with reason that experiments in exhaust-chambers and in balloons do not correspond with mountain-climbing unless time enough be allowed for readjustment between the outside pressure and the pressure inside the body and its parts, a time which is allowed in the case of climbing. Dr. Andrew Smith and other writers on caisson disease have shown that the danger of this occupation lies in the want of careful provision for the gradual increase and reduction of pressures; and that with such precautions extreme variations of pressure are borne with safety. The effect of reductions in oxygen values as pressure is gradually reduced may be counteracted by securing a sufficient supply of the gas in the caisson, or for the aeronaut. By such a precaution I gather that both Roy and Hepburn think that still higher mountains can be scaled, always supposing, that is, that the gas could be economically carried by the explorers. Mountain-sickness is felt more in sun than under cloud, and more in a trough, where the air feels stagnant, than in a wind. This effect of sun Roy attributes to a greater absorption of oxygen by melting snow; but Thomas found the sickness begin at a lower level on rock than on snow, and the explanation seems to be that the air is still more rarefied by the heat. A breeze, again, rapidly diffuses the expired air, removing it from the neighbourhood of the mouth.

Although I no longer think that *heart failure* is the immediate cause of

mountain-sickness, yet I am strongly of opinion that dilatation takes place to a greater extent in such exercises than is generally supposed; and in this opinion I am supported by Professor Roy. Into this subject I shall enter more fully in the section on "Overstrain of the Heart" in a later volume: meanwhile I have only to point out that the establishment of any degree of dilatation of the heart must form part of a vicious circle in which the evil is multiplied. This tendency to dilatation is shown in one or two of Conway's sphygmograms; but as these were necessarily taken at rest the effects of actual exertion on the heart are scarcely to be found in them. To take tracings during exertion is impossible, they are untrustworthy enough as it is; but, as Roy suggests, observations might be made with a flexible stethoscope by a sufficiently skilled observer.

On descent it would seem that discomfort is felt down to a lower level than that of its onset (namely, 13,000 feet); and this we should expect if at about 16,500 feet a greater relative absorption of oxygen has to be provided for. In some of Conway's sphygmograms the heart is slowed; this seems to point to vagus action sparing the heart; on the other hand, the ruling acceleration points both to fatigue and to the call of the tissues for more oxygen. The circulation of imperfectly oxygenated blood in the coronary arteries accounts for no small part of the cardiac embarrassment.

Symptoms.—The symptoms of mountain-sickness are best described in the words of the sufferers. Mr. Whymper says: "When we arrived at 16,664 feet we ourselves were in good condition, which was to be expected, as we had ridden most of the way; but in half an hour I found myself lying on my back along with the Carrels, placed *hors de combat*, and incapable of making the least exertion. . . . We were feverish, had intense headache, and were unable to satisfy our desire for air, except by breathing with open mouths. This naturally parched the throat, and produced a craving for drink which we were unable to satisfy, partly from difficulty in obtaining it, and partly from trouble in sipping it. Before a mouthful was down we were obliged to breathe and gasp again until our throats were as dry as ever. . . . We found it impossible to sustain life without every now and then giving spasmodic gulps, just like fishes when taken out of water." After some habituation Whymper says that the abiding symptoms were great lassitude and headache (and no doubt an accelerated rate of respiration which might become more automatic, or the bulb might become a little less sensitive to the venosity of the blood). Mr. Clinton Dent experienced precisely the same symptoms, including "feverishness," though he does not say, nor does Whymper, that the fever was measured by the thermometer. Now as to this symptom Sir Martin Conway's experience is decisive. That this man or that may feel "feverish" is probable, especially when we remember that fatigue products are circulating in the system; but that fever is not an essential symptom of mountain-sickness is proved by the accurate thermometrical observations of Conway, who says: "Bruce's temperature

and mine were both normal, notwithstanding that we plainly felt, and continued to feel, discomfort from the reduced atmospheric pressure." In a private letter now before me, Mr. Dent says that during a three weeks' residence at a height of 6000 feet with Mr. Buckmaster and another friend, in 1896, the mean bodily temperatures of the members of the party were always below the normal. Again, my own experiments (2), lasting over ten or twelve climbing days, showed that the mean of the bodily temperature was not depressed though the curve was rather acuter in its course. On one day, however, I discovered in myself a sudden drop like those recorded by M. Lortet, but I had no sensation of discomfort or weariness. In another passage of his narrative, Conway says—on the summit of Pioneer Peak, 23,000 feet: "We ceased to pant for breath the moment the need for exertion was withdrawn, and a delicious lassitude and forgetfulness of past labour supervened upon our overwrought frames. All felt weak and ill, like men just lifted from beds of sickness." These observers, the Gurkhas excepted, made no mention of nausea or vomiting, nor did any of the party suffer from the hæmorrhages which are recorded by Humboldt and others. It is a remarkable thing that many startling events which beset the early explorers have vanished on a greater familiarity with mountains. Nose-bleeding and other strange effects which befell the first scalers of Mont Blanc are not perceived by their successors. Hæmorrhages, such as nose-bleeding, gum-bleeding, and bloodshot eye, are likely, of course, to occur on rapid diminution of atmospheric pressure; and therefore ought perhaps to be included in the list of symptoms which may attack mountaineers; but under ordinary circumstances they seem to be absent. Nausea and vomiting, whether due to fatigue, poisons such as badly canned food, or an aggravation of ordinary indigestion, would not in any case form part of the characteristic symptoms of mountain-sickness, as these effects would occur in like manner on excessive exertion at any level; yet it may be important to remember that Roy and Cobbett (in some unpublished experiments) found the digestive tract in asphyxia to be anæmic. Conway's two Gurkhas vomited at 17,000 feet and 22,000 feet respectively; but for caste reasons these men were unable to modify their ordinary diet. Headache seems to be a constant symptom; and, as Dr. Hepburn says, may be explained on any hypothesis — cerebral anæmia, cerebral hyperæmia, etc.—as may be preferred by the individual commentator (7). Palpitation also is mentioned by all observers, and is familiar to all climbers. I once took three friends up the Piz Morteratsch (12,316) without guides, and two of them, one an active young lady, were so prostrate with palpitation, I feared the common duty of a guide to carry his tourist up the last 100 feet might be my lot. But I found that if the patient lay down for ten minutes the heart gradually fell to its proper rhythm, and by one or two repetitions of this device I got my friends well to the summit. It seemed clear, therefore, that the palpitation was rather a symptom of fatigue than of asphyxia, though it was aggravated no doubt by the rarefaction of the air.

Tinnitus, although mentioned by all climbers even at relatively low levels, is but a result of unequal pressures upon the tympanum; and, however disagreeable, is of secondary importance. The experience of the Schlagintweits confirms on the whole that of Whymper and of Conway. Hepburn thus sums up the proper symptoms of climbing at levels above 16,500 feet. *Acute*:—(*a*) increased respiration with spasmodic gulps; (*b*) incapability for exertion; (*c*) intense headache; (*d*) slight rise of temperature (? ED.). *Chronic*:—(*a*) lassitude and fatigue; (*b*) increased respiration on the slightest exertion. The symptoms of the chronic group are those which continuously oppressed Mr. Kennedy and Fischer in Nepaul (private letters to myself). Animals seem to be affected in the same way. To these chronic symptoms I think coldness of the limbs may be added, and to both classes palpitation of the heart and more or less vertigo; this last symptom may be due directly to diminished pressure, and if so, it probably belongs to the first or acute group.

T. CLIFFORD ALLBUTT.

REFERENCES

1. ALLBUTT, T. C. "On Overstrain of the Heart," *St. George's Hospital Report*, 1879.—2. *Idem. Proc. Royal Soc.* 1871.—3. BERT, PAUL. *La Pression barométrique.* Paris, 1877.—4. CONWAY, Sir W. M. *Climbing in the Himalayas*, vol. ii. Lond. 1894.—5. DENT, CLINTON T. "Physiological Effects of High Altitudes," *Geographical Journal*, January 1893.—6. HEPBURN, M. L. "Mal des montagnes," *St. Bartholomew's Hospital Reports*, vol. xxxi.—7. ROY and ADAMI. *Phil. Trans.* vol. clxxxiii. (1892).—8. ROY and SHERRINGTON. *Journal of Physiology*, vol. xl. p. 85.—9. SCHLAGINTWEIT, H. A. and R. *India and High Asia*, Lond. 1862, vol. ii. p. 484.—10. THOMAS. "Mountaineering in the Rockies," *Alpine Journal*, May 1894.—11. WHYMPER, E. *Great Andes of the Equator.* Lond. 1892.—12. ZUNTZ. "Experiments on Respiration on M. Rosa," *Berlin Meteorological Soc.* Feb. 4, 1896. The many excellent contributions of Dr. Marcet to this subject are well known, and need no special indications; but I will especially refer to his Croonian Lectures R. Coll. Phys. 1895, published by Churchill, and entitled *History of Respiration in Man.*

T. C. A.

NEUROSES OF THE STOMACH

AND OF OTHER PARTS OF THE ABDOMEN

Introduction.—When too readily I took upon myself to write the article on this subject I was little aware of the difficulty of the task I had assumed. Some fourteen years ago I delivered certain lectures upon Visceral Neuroses; at that time it was an easier task to carry the advancing knowledge of nervous diseases into other parts of clinical medicine. That in the multiform perturbations of the stomach and other viscera the nervous system is often primarily, always eminently concerned, is now a truism; then it was something new. This factor

in causation, then neglected, may now indeed be over-estimated; or at any rate insufficiently correlated with other factors. Yet Dr. Goodhart, in his lectures to the Harveian Society "On Common Neuroses," says of indigestion: "If I were going to write a book on indigestion I should first devote myself to a volume on diseases of the nervous system"; and again, "It is no great exaggeration to say that there are only two forms of indigestion: that produced by over-eating and drinking, and that due to a failure of nervous power." But I must replace Dr. Goodhart's book on the shelf lest I be tempted to substitute his graphic pages for my own, an exchange which, however pleasant for the reader, would be immoral for me.

In the integration of animal function the proper activities of all our organs have been so completely reduced to the governance of the nervous system, that in a sense it may be said that their every act, their every disorder, whether in defect or in excess, is a function of the nervous system. The whole store of strength in the nervous system may run low; or excessive demand in one part of the body may divert a part of the due supply of nervous energy from another part, as from the stomach. No organ lives on its own funds; the very existence of a nervous system means a give and take between the several organs of the body: now one is in full play, now another; and this alternation of departmental activities is rendered possible on the principle of greatest economy. Energy running to waste as regards a resting organ is turned to the uses of others in activity. Thus the several organs are members one of another; when one suffers, another sympathises: and in clinical medicine it is one of our most interesting studies to discover these intimacies, to detect not only the general dependence of each organ upon the whole system, but also the closer alliances between groups of two or more organs. To some extent, no doubt, the nervous system is the fly-wheel of the whole machine; by its storage and distribution the various pulsations of the several parts are integrated: but it seems likely that within this great integration there are subordinate alliances of particular parts, so that at first affection of one is felt rather by allied parts than universally. Broadly speaking, for instance, the parts of the circulatory, of the alimentary, and of the respiratory systems hang together in a special dependence; though when one or more members of any one of these systems, or sub-systems, is gravely affected, its defect will soon be felt beyond its own circle. Strictly speaking, it is scarcely possible for any part to fail or suffer without some echo throughout the microcosm; but there are inner circles of diffusion within certain spheres. In a sense, then, there can be no dyspepsia which is not associated with nervous affection, whether directly by way of the nerves themselves, or by way of an intermediate intoxication spread by the blood to the nervous parts: these parts then betray disorder within their own sphere, and by their connections with the stomach may intensify the original disorder, or, on the other hand, may bring about some conservative reaction of which we know little.

On consulting the last editions of the treatises on physiology by Foster and by Hermann I gather, from Hermann, that severance of the vagi arrests the secretion of the stomach or renders it abnormal; that irritation of these nerves excites the secretion, and that the older opinion that the food carried into the stomach excites the secretion, either mechanically or by but a short reflection from its own ganglia, was based upon a false belief that the vagi had nothing to do with the secretion. Hermann points out, indeed, and Foster likewise, that when, by occlusion of the œsophagus, food cannot enter the stomach, the presence of food in the mouth, or even the mere sight or smell of it, may provoke a lively secretion of gastric juice. Again, as Foster says, emotions of grief or anger may arrest the secretion. Foster states that when the vagi are cut the secretion is no longer provoked by the presence of food. The secretion set up by mere mechanical stimulation of the coats of the organ is, according to Foster, very scanty in quantity as compared with that which flows on the contact of food. He adds the remarkable and, to us, the important fact that the absorption of certain products derived from the portion of food first digested heightens the secretory activity of the gastric cells; a statement which rests on certain experiments by Heidenhain. The intermediate means may, however, be not the nervous system but the arterial. Our knowledge has been still further advanced by the recent researches of Pawlow, to which my attention has been drawn by Dr. Gaskell. A clear account of these researches is given by Dr. Ernest Starling in *Science Progress* for February 1896; I give the references to the original papers in my list, but I have not myself gone beyond Starling's account. A curious proof of the dependence of gastric secretions upon mental as well as upon physical conditions—upon fright, for instance, or upon narcotics—is adduced by Pawlow. Pawlow, by an ingenious and cautious method, divided the œsophagus of a dog so that the cross-sections of the tube were exposed, one in each corner of the wound. When the dog was itself again it was found, as I have said, that even the sight of meat promoted a flow of gastric juice, but always after an interval of five or six minutes, which period of latency seems to be occupied by the intermediate glandular processes. In thirty-five minutes the secretion would amount to 17·8 cm. Now, if the dog were teased with meat in such a way that he saw he was not to have it, the secretion of gastric juice did not occur or was arrested. This observation throws a strong light on the common experience of digestion in man. Some interesting experiments were made upon the gastric secretion which indicated that the hydrochloric acid is in loose but intimate association with the pepsine, and supported the belief that the acid is derived from the chloride of sodium in the blood. If with certain precautions the vagi were then divided in such a dog, the secretion of gastric juice wholly ceased. In three or four days dyspepsia would be manifest; food would lie in the stomach and putrefy, the dog would waste and die of the poisoning. But if, twenty-four hours after the division of the vagi, one of them were

withdrawn from the wound and its distal end stimulated, gastric juice would be secreted again after the usual interval of latency.

Concerning the senses of the stomach it seems that we know little or nothing; but that the stomach is possessed of a quasi-tactile sense, of a sense of temperature, and of pain is known well enough to the least experienced physician, and for our present purposes needs no corroboration. The ingestion of cordials, or of pleasantly warm liquids, is evidence of one kind of sensation; the symptoms of indigestion or of ulcer suffice to prove the sense of pain. What we wish to know is whether pain, whatever its nervous machinery, arrests gastric secretion; if so, morbid hyperæsthesia of the stomach, like emotions of the mind, may inhibit the formation of gastric juice. It is to be desired that by means of test meals in cases of gastric hyperæsthesia in a neurotic subject experimenters will work out this problem [*vide* Leube, p. 470]. On the other hand, anæsthesia of the stomach in its various degrees may, and probably does, arrest or rather prevent the secretion which should follow the ingestion of food; the food may not be recognised, as it were. Thus women who have little appreciation of the pleasures of the table may not only fall into a way of indifference to food, but the stomach may thus be imperfectly supplied with gastric juice. We often observe that articles of diet of less digestibility on the average may agree better with one who enjoys them than other diets, theoretically better, which are unwelcome. Thus sometimes even "cravings" may well be indulged. That a less digestible dinner eaten with relish and mirth is often better digested than a dinner dictated by the physician, and eaten in the solitude of the chamber, is well known. If depressing emotions diminish the values of the gastric juice, exhilaration probably increases them.

With the directly mechanical causes of gastric function we have nothing here to do; our survey is limited to the senses of temperature, of pain, and of that quasi-tactile sense in the organ by which certain foods are recognised, consciously or not, as grateful or ungrateful. We have seen that certain nervous influences govern secretion directly; so indirectly also other nerves control the vascular supply to the organ: of the precise concern of the latter in digestion, and of their relation to the direct nerves of secretion, we know little or nothing except that activity of the mucous membrane is associated with reddening of its surface. Whether in any disease the nerves of the blood-vessels are primarily affected, and so the functions of the stomach consequently, we do not know. We do know, however, that the stomach, like the heart, has to provide for its own nutrition; and, like the heart, often falls into the vicious circle of doing as ill for itself as for other parts of the body. Whether the nerves of the blood vessels protect or nurse the stomach when over-wrought, as the vagus nurses the heart, we do not know. I do not even mention the great sympathetic, a nervous supply which is too large and too widely distributed to be without important functions. But it is better to wait till our knowledge comes than to prate about vaso-motor and sympathetic nerves to cloak our ignorance. Again, like

the heart, the stomach is possessed of a certain tone; and, likewise, this tone is not constant in degree, but varies largely with circumstances and capacities. Tone must be largely under the influence of the nervous system; but certainly tone is often reduced by bad or deficient blood, whether the blood be simply poor or be charged with poison; poison, again, may be brewed by the stomach to its own injury, as, for instance, in dilatation of the cavity; or, as in the infective fevers, it may have other modes of origin. This argument does not take us altogether away from the nervous system; for probably many of these poisons act through the medium of this system. A stomach by nervous defect may lose tone, food may be delayed in the cavity, poisons may be brewed there, and these may not only taint the blood, but may also so impair the nervous parts that the stomach may suffer both primarily and secondarily from the nervous system. In certain cases of neurasthenia, or pseudo-neurasthenia, the stomach is the primary source of the departure from health; in others, if, indeed, the initiation of the morbid series lay with the nervous system, the secondary impairment of the stomach may become the central feature, and the alleged neurasthenia be due to bad products of digestion which, returning again to the stomach, depress it and its work still further: if so, a cure is to be obtained by way of gastric medication only; the stomach is the link in the vicious circle which has to be forged anew. Some poisons, such as tea, coffee, and tobacco, seem to have an immediate action upon the gastric nerves; and the list might be much extended. Some of these agents are discussed at length in other sections of this *System*.

Nor do we fail to find evidence, in the phenomena of particular diseases, of the direct dependence of the stomach upon the nervous system; in asthma we have a malady which often concerns the stomach almost as much as the lung; and the vomiting of cerebral disease is to be observed in every hospital. Seeing, then, that the nervous aspect of the functions of the stomach, whether normal or abnormal, is perhaps but another expression for the dynamic aspect of these functions—for at this moment we may disregard the sphere of consciousness in the matter—it is evident that the nervous element in the causation of gastric derangements is all-pervading; it is none the less evident, however, that to consider the neurotic factor alone would vitiate every argument on the subject. If nervous disorder finds its expression in terms of gastric juice, or in a relaxation of muscular tone, shall we not do better to discuss the nervous element in terms of these local results, in terms of tone and secretion as they are more positively manifested in the affected organ itself? Shall we not be able to deal more positively with the nearer than with the remoter links in the chain? The reply of the working physician is that the statical aspects are less positive than they appear: to work efficiently we must know processes as well as results, the initiation as well as the issue of change; and for practical men the dynamical oscillations may sometimes be even easier to recognise, if not to measure, than the statical—if, of course, we avert our attention for the moment from the grosser forms of disease.

For instance, a nervous disorder may and probably will affect other organs as well as the one under present consideration; if the disturbance begin in the nervous system, gusts of it will probably be observed in various regions, although perhaps in none can the resulting defects be directly perceived or measured. Again, if tone in its largest acceptation be something more than the mere preservation of the capacity of a hollow organ about its normal mean, and signify the preservation of that degree of mutual tension or polarity which, like that existing between the several members of the solar system, keeps all parts in balance and in mutual service, in this case a relaxation of tone might be more readily perceptible in the rocking of the whole system or in a slackening of polarity between particular parts than in variations of secretion and the like within the organs themselves, which might be hard to appreciate. Thus, for example, the practical physician will say that a nervous dyspeptic is easy enough of recognition: whatever we may say of the solidarity of organic changes, he presents certain general characters, in addition to the disorders of the stomach, which stamp him as a "neurotic," as one in whom nervous capacity is diminished, or nervous quality perverted. Not only so, but with a little care we may pick out in a rough way the cases in which the stomach seems itself to have been the origin of the malady from those in which the stomach has suffered secondarily or, at any rate, in association with other parts—such as the uterus, let us say, or the lungs. If the physician, by virtue of his knowledge of the world, recognises his patient as one in whom, to use a convenient expression, the spiritual activity is evidently greater than the physical capacity; and if such an one complain of neuralgia of the face, of fits of exhaustion, of sleeplessness, of irritability, of loss of self-control in little things, of palpitations perhaps, and of muscular restlessness; if he be lean and bloodless, if his demeanour be excitable and impulsive, and if withal he complain of dyspepsia, it is perhaps true to surmise that the nervous system is primarily at fault and first needs the physician's attention; although the patient may complain most loudly of his stomach and, with the curiosity of such persons, may inquire for bismuth, soda, gentian, arsenic and like "cures for indigestion." Or, again, an anæmic young woman, rather lethargic than vivacious, may come to the physician complaining of severe pain in or about the stomach, of vomiting, of fits of polyuria, of weakness or numbness of limbs, of tenderness of certain points on the spine; and he must decide whether her distress be primarily due to some gastric lesion or to neurotic perversion, a problem which can only be answered by a consideration of all the features of the person herself, as well as by the manner of the pain: to make the diagnosis by examining the stomach itself and all its secretions would be less easy and more fallacious. A third patient may present himself with gastric pain and with vomiting, but also with a recent squint, with pains in his legs and a disorder of gait; is such a case one of "nervous dyspepsia"? Not to multiply instances, I have said enough to show that though I admit that in all the acts of the organ the

nervous element may be intimately concerned, there is a proper place for a study of certain stomach disorders from a nervous point of view. The difference is one of degree or one of initiation; and the difficulty of writing this article lies in the danger of deviation on the one hand into primary dyspepsias, on the other into neurasthenia, hysteria, and other diseases of the nervous system, maladies to be dealt with in later chapters.

Causes.—It is chiefly by the causation of neuroses of the stomach that they are known. Take a simple instance: a vigorous young man of twenty-two goes out to Switzerland; he engages in a first-class excursion before he has got his second wind; he comes home to his inn after eighteen hours on foot, douches himself after the manner of his kind with cold water, and then falls upon a large supper which he probably washes down with new Swiss champagne. This done he rolls off to bed heavy and weary, but his sleep is brief: in the small hours of the morning he becomes aware that he is restless, burdened at the stomach, and sickly. If he vomit he is a lucky man; if he take an emetic he is a wise one: if he toss about all night between waking and dreaming he will feel "bilious" all next day, and out of sorts for more than a day. A smart attack of diarrhœa, a costly remedy, may leave him out of condition for a week. If he vomit, on the other hand, the food will come up half fermented, but much as he had eaten it; after the evacuation he will sleep soundly, and awake bright and fit for work again. In this case digestion had scarcely begun. Another time he will know better; he will take a warm bath and turn into bed with no more than a basin of soup and a thimbleful of brandy inside him, and he will eat his supper next morning. Now what is the meaning of this dyspepsia? There was nothing in the day's work to hurt the stomach directly—quite the contrary; the stomach failed because the nervous system was exhausted [*vide* p. 399].

Again, a keen man of business takes no rest; he drives himself and those about him at full speed: every disappointment, every mishap worries him and he becomes nervous, fretful, sleepless, and—dyspeptic.

A little boy or girl is born of anxious, nervous parents; his life is artificial, he walks in his sleep, he is very excitable at play, and after play is tired out; his lessons make his head ache; if he catches some infectious ailment he starts and twitches or has even a convulsion. Now if we examine his body we shall see that his legs are thin, but his belly tumid; his bowels are constipated; he takes little or no breakfast, and he is qualmish and whimsical at all his meals. Between meals he may take tempting morsels, but not enough to supply the wants of active growth. Moreover, now and again he has inexplicable attacks of vomiting which no care seems to prevent. In these cases, again, the nervous system is ill controlled or wanting in capacity, or both.

Finally, gastric disturbances, apparently due to the mediation of the nervous system, are wont to arise in sympathy with disturbances elsewhere, as, for example, in the uterus, in the ureter, in the gall-duct. These sympathetic gastric neuroses are well known, and I may be content to indicate this side of their pathology.

To say that "nervous dyspepsia" is exclusively a disease of middle-aged and harassed men and women is to erect a group of symptoms into a "type," and thus to hold them aloof from other groups of symptoms which, perhaps, without much superficial resemblance, are nevertheless of a like kind. This being so, I shall not make patterns of the disease, but deal with the symptoms severally, indicating the relations of each as far as possible.

Symptoms.—*Pain and other sensory perversities.*—"Every schoolboy knows" how the stomach can ache; and, on the other hand, the physician is too often witness of the pain of ulcer or malignant disease of the organ. In the aching of a stomach which resents an unwholesome morsel there is nothing abnormal; but in some persons the stomach aches badly however bland its contents, nay, when it is altogether empty. Such pain is called gastralgia, and it is often a matter of no little difficulty to distinguish this pain from the pain of simple ulcer or of cancer. A pure neuralgia, apart from irritants, is not common before adult years; but it may continue thenceforward with intervals of relief up to old age. In the gastralgia of old persons, in whose case a suspicion of malignant disease more easily finds its way into the mind of the physician, we note, perhaps invariably, a previous history of the former malady, or of others akin to it, which, although not contradicting the supposition of graver disease, may suggest a more hopeful state of things. In character the pain varies. In many cases it is a wearing or gnawing pain, which seems to be very hard to bear; in others it is a sinking rather, or a misery; in others it is acute or lancinating; and in the latter case the pain may radiate into the abdomen, into the loins, or, upwards, into the thoracic, cardiac, and intercostal regions. The pain of mere neuralgia—of neuralgia, that is, apart from local disorder of the stomach—does not usually penetrate to the interscapular region: I am far from saying that it never does, for I have at present one such case under my occasional care, but such a direction of pain increases the suspicion of ulcer. In mere neuralgia the pain is often relieved by pressure, even by strong pressure, as by leaning over the back of a chair; and though in not a few cases there is some tenderness on pressure, yet this is more diffused than in ulcer, the tender area of which is often so small as to be covered by the point of the finger; the tender area of gastralgia is usually at least as large as the hand, and the skin is often hyperæsthetic over a much larger surface. Points of definite tenderness have been mapped out for gastralgia as for other neuralgias; in gastralgia they have seemed to me hitherto to be inconstant, and in diagnosis useless.

If the attacks of pain occur independently of food, as for instance in the middle of the night, or before breakfast—a not uncommon hour for its recurrence—the diagnosis is easier than when the pain recurs, as not infrequently it does, immediately or soon after meals. Even when it occurs soon after meals, however, vomiting is often absent in cases of neuralgia: not that in neuralgia vomiting is by any means unknown; on the contrary, pain and vomiting recurring with some regularity after food are by no

means always significant of ulcer; they may be but signs of gastralgia. Indeed we cannot say that pain, vomiting and hæmatemesis are decisive against mere gastralgia: hysterical women have a way of producing blood without apparent breach of surface; and I recall more than one case in which this triad of symptoms meant no more than "functional" disease of the organ. Without post-mortem examination a positive diagnosis may be impossible; yet in the particular case there may be circumstances to create a strong opinion in favour of the lighter diagnosis,—if lighter it be, for even ulcer of the stomach is easier to cure than gastralgia with hysteria. The pain of over-acidity of the stomach and that of gastralgia may be much alike, but the former is more readily relieved by a sufficient dose of an alkali. Gastralgia is usually, but not always relieved by taking food; so, however, is over-acidity: but there is a very troublesome kind of gastralgia in which the ingestion of nourishment, even the blandest, is followed by torments. This kind of attack is commoner in neurotic young women; in them the dread of food becomes so besetting that the sufferer cuts it down to the lowest point, and may thus reduce herself to extreme emaciation. In these persons the pain is not quite like that of ulcer; it is more unreasonable; in ulcer to swallow a spoonful of gruel is not an instant torture: still there are milder cases of this kind of hyperæsthetic gastralgia in which the differential diagnosis cannot be made by the features of the pain taken alone, though the other features of the case usually suffice to determine a right conclusion. At the same time it must be remembered that in some cases the pain of the ulcer itself is associated with pains which are rather of the nature of gastralgia, as if the local disease awakened a gastralgia. This apparent combination of pains is not infrequent; and the physician will do well to bear in mind that the presence of pains having the characters of gastralgia does not exclude ulcer, the pains of which may indeed be less importunate, and more or less merged in the neuralgia. The pains of malignant disease are usually aggravated by food; but, whatever their behaviour, gastric pains awakened for the first time in a person at or beyond middle life must give rise to anxious suspicions. It is said, I do not know with how much truth, that the pain of an ulcerated surface is aggravated by a dose of alcohol, while the pain of gastralgia is relieved. It is certain that the ordinary restrictions of diet, which so much relieve other disorders of the stomach, do not relieve gastralgia; for, if Leube's observations be verified, the digestive powers of gastralgic patients (as ascertained by test meals) is not much deteriorated. In one case, however, by the syphon I discovered delay of digestion; and I think that in this malady there is a tendency to variation in the secretion of hydrochloric acid—generally in the direction of insufficiency, very rarely in that of excess. We have already surmised that some reflex interference with the activity of the gland cells is to be expected [p. 465].

Pain, again, is left sometimes as a legacy by foregone disease, such as a gastritis which was attended by pain. In two cases of recurrent ulceration I came to the opinion that subsequent or intermediate returns

of pain were not always due to a return of the ulceration itself; in some phases, at any rate, the pain was an echo of the distress of the past disease. In one lady, now approaching the menopause, I have witnessed three or four attacks of simple gastric ulcer with all the classical symptoms; but between the definite attacks there have been times of alarm in which the pain was probably but neuralgic. I admit that certainty is impossible under ordinary circumstances; but, having carefully watched these two cases cited for many years, I give my opinion with some confidence. The disposition of gastralgia to increase about the menstrual period may be a point of difference. To make the distinction is of obvious importance, both in respect of prognosis and treatment. Another difficulty may lie in the distinction between gastralgia and the true or the false angina pectoris. Of the former alternative I need say nothing; the circumstances of the individual case will surely guide the observer. But between the false angina and gastralgia the distinction may be difficult; though indeed it may not be of much practical importance. The agitation, the air hunger, and the paroxysmal modes of recurrence will probably suffice to lead to a right interpretation of an anginoid attack; and, conversely, abdominal cramps, and abdominal pulse, and other features may incline our opinion to gastralgia; but it is to be remembered that the actual seat of the pains is as little known in the one disease as in the other, and that the two may well overlap, or may be associated severally or together with other abdominal neuralgias. To add to our perplexity a remarkable alliance between gastralgia and aortic regurgitation has been pointed out by several writers, by myself among others. No definite explanation of the coincidence has been proposed [*vide* art. "Aortic Disease of the Heart" in a later volume].

The gastric crises which are a part of spinal disease must be diagnosed on other evidence; the pain in itself may not be decisive, but its paroxysmal character and intervals of absence are unlike gastralgia, which, if not continuous, is more persistent. The degree of pain in gastralgia may be quite as great as in tabes dorsalis; indeed, it is sometimes so violent, both in itself and in the cramps which attend it, as to throw the sufferer into a collapse. I believe I once saw such a collapse prove fatal in a highly neurotic woman, long a victim to abdominal neuralgia; her pulse failed, her limbs and face grew cold, and she could not be brought round: the absence of a necropsy precludes me from speaking confidently. But I once saw such a death follow the passage of a gallstone in a healthy young woman; in this case an autopsy was made, and with the exception of the stone, which was found in the uninjured common duct near its entrance into the gut, the body was perfectly healthy. We must not assume, therefore, that mere abdominal pain cannot be fatal.

On the other hand, the pain of gastralgia may be very slight; often it amounts to no more than a sense of weight or fulness in the pit of the stomach, or a restless uneasiness or sinking. Heat rather than pain proper is sometimes the complaint of nervous dyspeptics. One patient—a middle-aged serious man—described this heat to me as burning all

down his breast-bone and epigastrium and even into the abdomen; it was like "liquid fire," he said; and it was made worse by food even of the simplest kind. This symptom, not uncommon in more moderate degrees, I regarded as due to hyperæsthesia, and not to Prout's hyperchlorhydria. There was no evidence of over-acidity, nor was the heat relieved by large experimental doses of alkali. Light sponge-cakes did best for this man; and he could take eggs in weak tea without much aggravation of his sufferings.

Gastric distress in neurotic persons, then, is not always pain properly so-called; often it consists in perverted organic sensations which may be no less hard to bear. Of these perhaps the commonest is the sense of sinking, which seems to be peculiarly distressing; this distress is not uncommon in intelligent and sensible persons. Such an one describes the sensation, referred to the epigastrium and upper abdomen, as one of utter exhaustion; the voice becomes weak and slow, and the face turns gray; but the pulse does not falter, nor does the attack feel like a faint, or lead to it. Sometimes this sensation is due to the use of tea by persons intolerant of it; but it may occur independently of any such agent. Food does not relieve it much, nor indeed is alcohol a very rapid means of comfort. A cup of hot beef tea well spiced answers as well as anything else, and is at least harmless; patients who suffer thus are liable also to false hunger, an acute and depressing hunger rather than a normal appetite: food soon brings satiety, but not much relief. Mid forenoon and late afternoon are the usual times for this discomfort; or it may come on in the middle of the night.

Bulimia is a rarer phenomenon, and I am not sure that this perversion is very characteristic of gastralgia, though it seems to occur in neurotic subjects. Some of these cases are of astounding severity. One I remember in a young man of some thirty years of age; in him the craving was prodigious. Not only did the patient eat enormously during the day, but he could not pass the night without gorging himself more and more. His landlady, a kindly soul who grudged him nothing, was unable to repress a tear when she told us that he would call for as many as thirty huge sandwiches between night and morning, sandwiches two to the slice of the quartern loaf; of these he would take ten well lined, each of them, with ham or beef, and often would ring once or twice for another plateful before morning. The poor woman had been wont to keep the patient at a price; now she bore a divided mind between anxiety lest she should pine her pensioner and lest he should be the ruin of her. In less degrees, scarcely to be called bulimia, this craving is not uncommon; and it is alleged by some observers that it depends upon an affection, temporary or organic, of the pneumogastric nerve. In one case a neuroma was discovered upon one pneumogastric trunk.

Vomiting.—Ever since as a novice I read Watson's *Lectures* I have been on the watch for cases of the malady clearly described by many writers, and very vividly by Watson, under the name of pyrosis. Morning vomiting or puking is common enough, especially in topers; so it is

in those afflicted with renal and other diseases, and vomiting is far from uncommon in neurotics who take no excess of food or drink. But this is not pyrosis, which must have become extinct, for the description was obviously taken from nature. I have asked my out-patients over and over again to describe their morning qualms or vomiting, and in private practice I have inquired after similar symptoms, but of true specimens of the classical pyrosis I have secured very few for my collection; race, or habits, or luck seem to have been against me.[1] One good case which I published in *Visceral Neuroses* (p. 44) was clearly of neurotic origin. The vomiting of gastralgia may occur at any time of the twenty-four hours; for the most part it is excited by food, which in these cases must be reduced in quantity. This vomiting occurs chiefly, but by no means exclusively, in women, and these women young; it may also occur in males, but always, I think, in young adults or children. Dr. Sidney Martin gives a case of the kind in a young woman who vomited every meal for intermittent periods during three years. The curious thing is that these patients often keep fairly plump in spite of their waste of good food; no doubt some considerable part of it is retained. Martin says that these patients are always neurasthenic or hysterical; yet I have seen many cases which could not thus be classed, unless we class thus all cases in which there is a morbid reflex irritability of any organ, with or without other symptoms of either of these more general maladies. The cases are very intractable, especially if treated at home, and the patients, if in comfortable circumstances, may soon be nursed into hysteria; but unfortunately the affection "functional vomiting" is not confined to the well-to-do, and breaks out also in poor, hard-working women, who are reduced to great straits by it. In them, no doubt, the causes are over-work and ill sustenance, such as a sparse and monotonous diet of bread and tea. Watson describes a case after scarlet fever in a girl of sixteen; she was cured by restricting the diet, not to frequent small quantities, but to one meal a day. He truly points out that in these cases nausea is infrequent. Some dilatation of the stomach may be present, but, when present, it is a subordinate and transient feature, as I know from the use of the syphon in many such cases. Regurgitation and even rumination of the upcast food are curious features of some cases of nervously irritable stomachs. The habit, for such I had almost called it, is not necessarily associated either with pain or vomiting.

Flatulence, which may or may not be associated with pain, is a remarkable "neurosis" of the stomach. It generally rises in storms, with such volumes of noise and gust that one wonders whence it can come. Curiously enough distension of the epigastric region stands in no direct relation to the flatulence; the two events often concur, but the sense of distension, which may be so great as to oblige the sufferer to undress, may not be associated with enlargement of the measurable waist; under these latter circumstances the wind may be tremendous nevertheless. To pursue this matter far would lead me into the sphere of hysteria,

[1] Dr. Lauder Brunton seems to explain the ambiguity, p. 387.

especially were I to treat of wind swallowed, as often it is, and afterwards regurgitated. In all these cases, as Graves pointed out, there seems to be some source of flatus other than decomposition of food. That air may be poured into the alimentary canal from its own walls is possible, I can neither aver nor deny it; and, if this be so, it may explain some of these strange phenomena. One sensible, hard-working professional man tells me that not unusually he wakes in the small hours of the morning, sits up in bed, and rolls up wind, belching it forth boisterously for many minutes. After some repetitions of these performances he lies down to sleep again. I need scarcely say that he is a light sleeper at the best; that he is a spare, sallow, restless man; and that he is a nervous dyspeptic of the classical kind. With the flatulence much disturbance of the heart is wont to be associated; but to follow this symptom beyond its mechanical causes would lead us into a general discussion of hysteria and neurasthenia. Dr. Goodhart remarks that the neurotic flatulence of man is more commonly in the stomach, of woman in the bowels. The vented gases are, as a rule, inoffensive; if offensive, there is more than gastric neurosis in the case. I ought perhaps here to except those obscure cases in which *sulphuretted hydrogen* is discharged from the stomach, often in such quantity as to make the whole atmosphere of the patient offensive. The only recent instance of this kind in my experience was in a young woman, undoubtedly neurotic, and neurotic only [*vide* art. "Dilatation of the Stomach," p. 498].

Anorexia nervosa.—Under the convenient title of anæsthesia I have referred to a state of stomach in which the natural sensation of hunger is diminished or even absent. Such cases are met with, always perhaps, in neurotic women, but not necessarily associated with other symptoms of hysteria; of neurasthenia I will say nothing, for this malady cannot persist without weakening all the systems of the body. To these not uncommon cases, already described by other observers including myself, Sir William Gull gave the name of anorexia nervosa, and the name is a good one. When for many months food has been taken in utterly inadequate quantities, neurasthenia (in its etymological sense) must follow, and asthenia of all and any other systems of the emaciated body; yet it is a remarkable character of these patients that they continue capable of occupations, interests, and even of efforts which, if not in themselves extraordinary, are at least astonishing in such frailty. A young woman thus afflicted, her clothes scarcely hanging together on her anatomy, her pulse slow and slack, her temperature two degrees below the normal mean, her bowels closed, her hair like that of a corpse dry and lustreless, her face and limbs ashy and cold, her hollow eyes the only vivid thing about her—this wan creature, whose daily food might lie on a crown piece, will be busy with mothers' meetings, with little sisters' frocks, with university extension, and with what you please else of unselfish effort, yet on what funds God only knows. At meal times her mother may cry, her father may storm, her friends may banter, and the cheerful reply never fails, that she has eaten amply;

or, if not, that she can eat no more. Every physician of experience will say that this is no overcharged picture. Many young women indeed, of ardent and self-forgetful nature, and happily there are many such, go near to fall into this state. Eating seems to them a crass matter; meat, even the smell of it, makes them sick; they declare that they eat as much as other people: nay, pathetically enough, these very ascetics are often beset by the notion that some other member of the family circle is not eating enough. Gradually the appetite neglected may begin to disappear, perhaps for want of elements in the gastric secretions which are dependent on demand; and so by degrees, and without the patient's own consciousness, the body is painlessly pined. It has been stated that a superficial cutaneous area of anæsthesia, corresponding to the area of the stomach, is characteristic of anorexia (9, 10). My own clinical experience does not bear this out, nor, I believe, the researches of Dr. Henry Head. Such anæsthetic patches probably form but a part of the distribution of hysterical anæsthesia. Nor have I noted dilatation of the stomach in anorexia nervosa; in extreme cases it may appear, perhaps, but the stomach is usually small: squelchy sounds on manipulation are not certain signs of ectasis. In minor degrees this state of appetite may be recognised more or less in men also; and at all ages short of old age. Such a failure in old persons is of a different nature, and means at least an atrophy, whether of the stomach itself or of other parts, which is no mere neurosis. With the state of anorexia nervosa gastralgia, or hyperæsthesia of the stomach, may or may not be associated; cases of the former are often seen without the latter. Vomiting, which seems to belong rather to another chain of gastric neuroses, is usually absent. It is not unlikely that numbness of the stomach in the hypochondriacal or melancholy may give rise to the delusion of non-existent stomach.

Motor disorders.—Of symptoms concerning the motor functions of the stomach in gastralgia there is not much to say. To loss of muscular inhibition may be attributed the manifold gurglings and other noises to be heard only too often in such persons, often without pain. In society these murmurings, and other audible noises of the gastric machinery, are often very untimely, and our aid may be sought in the removal of them. This is no easy task; they can only be dealt with on general principles. It is too often alleged that in such stomachs the organ or its muscle is in such an irritable fuss that the food is hurried with undue rapidity into the intestines, and proper digestion thus prevented. This may be so; the stools of such patients must be inspected in order to see whether the food passes through imperfectly digested. However, so far as I have made any investigations which deserve the name of scientific, delay rather than acceleration was discovered; motor disorder is usually, I believe, in the direction of defect rather than excess, though no doubt, if excited by abnormal contents, the gastric muscle has its times of resentment. I have already said that in these fretful stomachs a false splash, by which I mean a splash which might be taken for

the splash of dilatation, is often to be obtained. To the expert physician the sounds are not closely alike; that of gastralgia is a squelch, that of dilatation is a slopping sound, and the toss of the gastric contents in a baggy stomach may seem almost or quite palpable [*vide* art. "Dilatation of Stomach," p. 505]. It is not unlikely that during cramping pain the stomach is spasmodically contracted. Certainly on inspection the epigastrium is often seen retracted, and the recti muscles of the abdomen very tight; there may be the same conditions within.

Of the influence that nervous irritation or arrest may have upon the *secretions* of the stomach I have said enough, perhaps, in the introduction to this chapter.

When we turn to consider *symptoms other than gastric* which are associated with gastralgia and its allies we are in danger, as I have already said, of travelling out of the field, and of trespassing upon that of neurasthenia or of hysteria, of hypochondriasis or of melancholia, maladies with which disturbances of the stomach are often associated, whether as cause, consequence, or coincidence. Thus symptoms of what are called a "predisposing" kind must be omitted in this place. But there are symptoms which may be regarded as subsidiary to or even consequent upon gastric neuroses, which must be briefly enumerated. In severe gastralgia the pulse may be retarded and lowered in pressure, the face and limbs may grow cold, even the teeth may chatter. In the lower part of the alimentary canal colics, windy tumidities, and occasionally phantom tumours arise, and therewith light diarrhœas are often associated. In the bowels, moreover, the gastric drama is often re-enacted. In the bowels pain as severe as that of the stomach may arise, with borborygmi and hyperæsthesia of the surface which, together with the tenseness of the abdominal walls, may arouse suspicions of inflammation. Trousseau says with truth that colalgia may be so closely associated with gastralgia as to be indistinguishable from it.

Of symptoms still further afield may be mentioned polyuria—which does not necessarily signify hysteria, vertigo, panting, palpitation, throbbing of the abdominal aorta, and yawning sluggishness after meals. To say that these symptoms, or some of them, prove the existence of hysteria is to say that all neurotic perversions are of the nature of hysteria.

Finally, many gastralgics are also liable to asthma, and again to eczema. In a few cases gastralgia in later life turns even to the true angina pectoris. These affinities are often to be recognised in the course of private practice, where careful evidence of past illnesses and of family pathological pedigrees is to be obtained.

Diagnosis.—In the course of the analysis of symptoms I have incidentally concerned myself with certain points of diagnosis, for instance with the distinction between gastralgia and ulcer, to which I need not return. If ulcer be not in question the greatest difficulty will be found in distinguishing between gastralgia and gall-stones. At a given moment the differential diagnosis between neuralgia and gall-stone may be impossible, and, at any time, it must often be inferred from the history of the case;

I am contemplating, of course, those cases of gall-stone colic only in which jaundice is absent—cases far more numerous than is generally supposed. We are not ashamed to admit that it is often impossible to decide at once between the one disorder and the other; still, in most cases the occasional occurrence of seizures in the midst of apparent health, and the absence of neurotic features, will enable us to make a true diagnosis. The difficult cases are those in which gall-stone pain becomes almost chronic, yet without jaundice, and in which, by the repetition of attacks, the patient is unnerved. I need not say that gall-stones may properly be suspected in young and clear-skinned persons as well as in the middle-aged. By the immediate characters of the pain I think that no diagnosis is to be made, at any rate not always. In gastralgia shivering and even chattering of the teeth, without elevation of temperature, are not uncommon. Another serious difficulty in diagnosis lies between neuroses, gastralgia especially, and malignant disease of the stomach; such problems arise in patients at or after fifty years of age. Gastralgia is by no means unknown in persons advanced in life, and indeed in men and women, not perhaps highly neurotic, and certainly not hysterical or melancholic, but of anxious or irritable habit, who are reduced in health by overwork or harass, and who have fallen into that state of indefinite ailment which so often appears and departs in the time of transition from manhood to age. Under these circumstances gastralgia or unaccountable vomiting may appear for the first time and without previous history of manifest neuroses. If a man or woman of fifty-five to sixty-five years of age, losing the bloom of maturity and falling somewhat in flesh and strength and appetite, consult a physician for pain in the region of the stomach, diagnosis may be very difficult, and on one visit impossible. The patient must be put to bed, closely watched, and very carefully examined on every visit; for a thickening of inner parts may be imperceptible on one occasion and perceptible on another. If, thus watched and carefully treated, the patient gain weight malignant disease may nevertheless be present; and perhaps no final assurance of safety can be given until a month or six weeks of supervision have elapsed; indeed the reader must not forget that pains of gastralgic character, and even of gastralgic nature, may arise under the irritation of local disease. An instance of gastralgia due to the dragging of an old adhesion is given in vol. ii. p. 887, and the reader is referred also to the article on "Enteroptosis" in this volume. Between gastroenteralgia and peritonitis a doubt may arise. Great superficial tenderness and severe pain within, together with a nipped expression of face and small pulse, may combine to the physician's embarrassment. The thermometer is of little use here. The history of the patient and of the attack will scarcely fail to give some help in diagnosis, and unimpeded action of the diaphragm would be a favourable sign, if, for example, perforating ulcer were suspected. Hiccough, vomiting, and meteorism, if present in any degree, will scarcely simulate those of peritonitis. In a word, no great difficulty is likely to occur.

Finally, in all cases of pain about the waist and abdomen, the knee-

jerks must be tested and the vertebral column closely examined. If the knee-jerks be absent, the diagnosis of neuralgia in its narrower sense must at any rate be postponed.

Treatment.—The treatment of gastric neuroses is of two kinds or purposes. Immediate relief is urgently needed in bad cases; in all cases general treatment is needed to cure both local distress and the state of system to which the malady is due. First let us suppose that the gastralgia appears in a young woman whose general condition is not one to cause much anxiety. She may be anæmic, in which case her general condition will not be forgotten. Iron alone may bring relief; but if not, a few drops of Fowler's solution, carefully used, will pretty surely secure this end. It is needless to say that the diet and mode of life must be carefully ordered. Next let us suppose the case to be in an older person, of either sex, of neurotic habit, and reduced by pain, refusal of food, or adverse circumstances. The first thing to be done is to put the patient to bed for a week; no excuses arising from mere restlessness are to be admitted. Many a bad case of gastralgia, as of other neuralgia, has been cured by a fortnight or three weeks in bed with careful management of diet; the warmth, rest, and seclusion from affairs, the coaxing of bland food into the weary stomach, and the administration of a little hydrocyanic acid being the chief agents of relief. If massage can be added, light at first and increased as the strength will bear it, the recovery will be facilitated. If after the first two days in bed the pain still be troublesome, let opium be given. It is better for obvious reasons to avoid the hypodermic syringe; and, fortunately, in these cases no means answer better than small doses of the solid drug administered in a pill, of the contents of which the patient may well be ignorant. I have found Dover's powder in doses of three or five grains very suitable; small pills or cachets are often retained when liquids are vomited, and I think the gradual solution of the pill is an advantage. In the pill, or otherwise, some means may be used to avert constipation. Cannabis indica is recommended by many authors, but opium is far more trustworthy, and, if kept under the control of the physician, may be used for some days, or even for two or three weeks, without ill consequences. Cocaine is not more useful, and has disadvantages of its own. As the digestive act itself is, by the nature of the case, supposed to be fairly normal, no acids, alkalies, bitters, and the like are requisite. Nor in my experience is rectal feeding, so useful in many stomach diseases, required in these; although a clyster of water as hot as it can be borne may relieve the pain. For the same reason I do not trouble myself much with pepsine, pancreatine, or pre-digested foods. The stomach can deal with tender and bland articles of diet well cooked and divided and given in small quantities; and they are far more acceptable to the patient and enticing to his stomach than peptonised foods. Indeed, too pedantic a rule of diet in these cases is to be deprecated; even whims must be regarded, and occasionally welcomed. Of other remedies, warmth and even mild counter-irritation to the epigastrium are profitable. After a

few days silver, either as the nitrate or the oxide in pills, will be of service in combination with the opium; or, if this drug be no longer necessary, alone. As the gastralgia subsides the patient must be re-edified in the usual way. Arsenic must not be forgotten as one of the best remedies for chronic gastralgia; but during the acuter phases opium is the one drug of real service. The value of arsenic in asthma, in eczema, and even in angina pectoris, probably depends on the same virtue, whatever it may be. If there be such a thing as malarial gastralgia, of which I can find no positive evidence, quinine must be the means of cure. I have not found strychnine of any great service in any form of the malady. That the epigastric pains of gout are gastralgic in the sense in which we are now using the name is improbable; if gout or hysteria be concerned in the matter, the treatment appropriate to these diseases must be applied. In the chapter on "Dilatation of the Stomach" (p. 505) I refer to pulmonary gymnastics as very important in certain cases in which irritable stomach or bowel is associated with defective expansion of the chest.

For anorexia nervosa there is but one cure, namely, bed, and feeding by a judicious nurse not of the family. I should say that the cure of such a case ought not to be attempted at home, and that the patient should under all circumstances be removed to the care of strangers, were it not that I have succeeded more than once in compassing a cure at home. But in any case a trained nurse is indispensable, and it is also indispensable that she have her own way undisturbed by the interferences and opinions of the family and friends of the patient. If this cannot be secured the physician will not desert his post, but he will disclaim all responsibility for the failure which will probably be his portion.

Of all these cases the most difficult are those of neurotic vomiting. These patients are better removed from home, but removal is not the almost certain cure that it is in nervous anorexia. Were it easy to prescribe the means of cure there would be less of the difficulty which I have indicated; food, however judiciously administered, too often returns, whether it be given in the smallest doses or given, on Watson's plan, in rather larger quantity once in twenty-four hours. The irritability of the stomach prevents the very means we desire to use. Massage without generous diet does but exhaust the patient; lavage, which I have carefully tried, is of little use; drugs are rejected. On the whole, the best means are rectal feeding, with the use of sedatives to calm the stomach and establish a gradual tolerance of food. The hypodermic use of morphia, as we know, is to be avoided if possible; moreover, in many of these cases, as its first effects pass off, it sets up some additional nausea. I have found most advantage from the bromides with hydrocyanic acid, or if this fail, with chloralamide. The latter combination, if administered in small and repeated quantities, often soothes the irritable coats, and food is retained. "Chlorobrom" may be found effectual. Of oxalate of cerium I have no great experience; but if it is to be of any value, it must be used in much larger doses than those usually ordered, namely, in 5 to 10-grain doses. Bismuth, in small doses of 10 to 15 grains, is valueless, or nearly

so; it has been used recently in doses of $\frac{1}{4}$ to $\frac{1}{2}$ an ounce mixed with a large quantity of water as an irrigation, but I have no experience of the plan. Of arsenic I can say more; with drop or half-drop doses in half a teaspoonful of water I have seen the stomach quieted in not a few severe cases of neurotic vomiting. It is best that these patients, if not kept altogether to bed, should lie down after food, with perhaps a hot bottle to the epigastrium; and as nausea or disposition to vomit comes on, the patient, in spite of efforts to the contrary, must be dissuaded from raising his head. After trial of all such means without success the patient may take a quick turn and recover of himself; perhaps, indeed, this is the issue of most cases of neurotic vomiting.

The only remark I need make about diet is that alcohol is a dangerous remedy to recommend to neurotic persons; fortunately, they do not find much benefit from it. In moderate quantities, however, with meals only, it may be valuable during the worst phases of health, and may be pretermitted on complete convalescence. The effects of tea, coffee, and tobacco on these patients should be carefully watched, they or one or other of them may be injurious; and the odd reactions of some persons to certain foods, such as eggs or shellfish, must not be forgotten.

Other abdominal neuralgias.—I find a difficulty in dealing with some other abdominal neuralgias, which undoubtedly exist, on account of the difficulties in their diagnosis. Although, on the one hand, the subsequent history of a patient in whom such pains have been set down as neuralgic may establish the correctness of the opinion, yet certainty in many of them cannot be predicated until after the lapse of many years. For in some the existence of a false band, of a latent calculus of this kind or that, of some disordered and acrimonious secretion, of an ulcer, of some unsuspected baneful article of diet, of latent cancer, or indeed of a benign growth or aneurysm causing pressure on the branch of a nerve, may originate pain which it would be an abuse of terms to call neuralgic, at any rate in the clinical sense. Again, we may have to deal with that source of wearisome pain which is attributed by M. Glenard to a fall of the viscera, a subject to be considered in the next volume, as regards the kidney by Professor Macalister, and as regards other parts by Mr. Treves. There is little doubt that persistent uterine pain is often set up by dragging of this kind in neuralgic subjects.

Perhaps enteroptosis in its lesser degrees does not cause pain in persons whose nervous system is stable; yet as, even in the cases of nervous instability, the distress is dependent on mechanical causes, and consequently its treatment on mechanical means, they are not conveniently discussed in this place. As I have said in respect of gastralgia so of other neuralgias of abdominal viscera, it may be that adhesion of an organ such as the gall-bladder to the parietes may occasion considerable pain which may have a paroxysmal character.

Still, all deductions made, it seems probable that there are cases in which visceral pain is purely neuralgic in nature; it is either

independent of local disease, or, if called up by some transient tides of local disorder, these bear so slight a proportion to the pain that they may be almost neglected; I say "almost," because the thesis might be sustained that all neuralgias depend upon local determinants, however fugitive and incalculable. That a diagnosis of abdominal neuralgia may, however, be correct sometimes was perhaps sufficiently proved by the case of a lady who called upon me a few days before these words were written. She reminded me that she had consulted me twenty years before for attacks of violent pain in the region of the liver; that in consultation with her own medical man we had decided that her pain was not dependent on gall-stone or local troubles, but was purely neuralgic (the grounds of the decision I cannot now give). She added that there was no doubt of the truth of the opinion, as the attacks had gradually diminished in frequency as her general health became stronger, but that she still had about two attacks a year, usually after some chill or other depressing cause.

Hepatalgia.—In my book on Visceral Neuroses I expressed my opinion that "hepatalgia," whether the pain be actually in the liver itself or in its appendages, may be a true neuralgia, and may exist apart from such local causes as we are accustomed to enumerate; and in support of this opinion I am able to quote Pariser, who, in commenting upon seven cases of "nervous hepatic colic," says: "This malady is usually mistaken for gall-stone colic, and in one case an operation was performed under this erroneous impression. Furbringer thinks that in hepatic neuralgia the most intense pain is localised in the liver, and is not radiating, and although this is true in many cases, exceptions do occur." The attacks, Pariser says, last from a few minutes to a few hours and, as in biliary colic, may end in vomiting. He defines the disease as visceral neurosis with a neurasthenic or hysterical basis, and treats it accordingly. To distinguish mere hepatalgia from gall-stone must often be impossible, even in patients of neurotic history; for the neurotic habit gives no immunity from gall-stone. In hepatalgia the liver is not enlarged, but it may be tender.

Enteralgia is not an infrequent disease, and is a very terrible one; it is perhaps the most terrible of all the neuralgias. Whether the pain lie in the bowel itself or elsewhere is a question which I have never been able to answer, even to my own satisfaction. It is usually a piercing, agonising and prostrating pain leading soon to symptoms of the incipient collapse which is at hand in all abdominal neuralgias, presumably because they tend to promote dilatation of the abdominal veins. Somewhat unlike the other abdominal neuralgias, it is perhaps commoner in men than in women, or at least as common. In my book I have recorded several cases in men which I need not repeat. Like gastralgia it is often associated with spinal neuralgia, so that the patient is racked by pain along the course of the associated spinal nerves as well as within the abdomen itself; therefore knee-jerks and the vertebral column must be examined as suspiciously as in gastralgia. In one of my cases a lady, for

some years subject to gastralgia, fell a victim later to the worse evil of enteralgia.

In not a few instances in women there may be a difficulty in discriminating between enteralgia and pelvic neuralgia; indeed, the confusion may at times be real, for neuralgia of the pelvic parts in women may precede or accompany enteralgia. It is pretty certain that enteralgia is associated more definitely with gout than are the other abdominal pains, though no doubt there are many cases of enteralgia which own no such cause. For instance, a gentleman I saw some years ago with Mr. Holmes of Leeds presented the following series of nervous miseries: first, he had suffered from cervico-brachial neuralgia with hyperæsthesia of the scalp with Valleix' points; at a later date he suffered from insomnia; later, again, from a strange sensitiveness of the skin to cold—the slightest draught, such even as the wafting of the leaves of his ledger, being painful to him; then periodic coryzas, with extreme defluxions and asthmatic dyspnœa, fell upon him; and, finally, with no less vehemence, he took to enteralgia. He was much benefited by a long rest with the systematic use of sea-water baths. I have never found electricity of much service in any of these cases; it gives no immediate relief, and perhaps we are not faithful and patient enough to persist in its use.

In the diagnosis of enteralgia we are less embarrassed by the alternative of gall-stone, the resemblance to this colic is not great; enteralgia usually begins near the navel, is more stabbing than gall-stone, and takes larger excursions not only about the belly, but, as I have said, in the courses of the neighbouring spinal nerves. The belly may be blown out, even with open bowels; or again, and more frequently, it is retracted and tense. The behaviour of the bowels is often irregular, irritable or obstinate; membranoid shreds, if present, must not be overlooked [*vide* Membranous Colitis, art. "Diseases of the Colon," in this volume]. One malady, however, there is which must not be mistaken for enteralgia proper; namely, the pain at the hepatic flexure of the colon which haunts the victims of melancholia or hypochondria. This pain is never acute, it is rather of a wearing and depressing character; moreover, it needs quite different treatment. The blue pill and black draught which relieve the hypochondriacal pain would greatly aggravate that of enteralgia. Arsenic is not the sure friend in the other visceral neuralgias that it is in gastralgia, but it is not without value. Belladonna is of some use, but cannot achieve much in severe attacks. I need not emphasise again the warning against the risk of permitting any of these abdominal neuralgiacs themselves to get hold of opium in any of its forms; the relief is as certain as the pain they suffer is dreadful, but the subsequent abandonment to the drug means physical and moral destruction. [*Vide* art. "Morphinism."]

If, then, we cannot deny them the gift of opium from time to time, we shall counsel these sufferers to submit patiently, persistently, and hopefully to a methodical course of diet, hygiene, and medicine, which, by removing the causes that in each case may appear to be operative, and by raising the state of the nervous system and of the blood, and the

nutrition of the tissues to the highest point, will place them above the reach of these perturbations. I should remind the reader that evidence of lead must be carefully sought for in all cases of enteralgia; and scrupulous search must be made for the minutest umbilical hernia.

Nephralgia is perhaps the commonest of the abdominal neuroses, and the one of which we know most; seeing that on occasions not a few a neuralgic kidney has been opened in vain in quest of stone. Thus that one kidney may be the seat of a grievous pain of a "functional" kind supports the hypothesis of the possibility of pure neuralgia in other abdominal viscera, such as the liver and stomach; though it cannot be definitely asserted that the pain actually lies in the kidney itself, its apparent seat. To distinguish the renal colic of gravel from mere nephralgia may be practicable if sufficient attention be given to the urinary deposits; but to distinguish the pain due to a solitary stone is too often impossible. Nausea or vomiting may coincide with either affection, and even the appearance of blood in the urine is no crucial fact, strange as this may seem; many an aching kidney has been opened on the indication of hæmaturia and found empty. Some authors appear to find satisfaction in calling such hæmorrhage "angioneurotic." Dr. Goodhart (p. 119) reports a remarkable case of this kind in which a necropsy was made with negative results; and another, in which operation was repeatedly performed, is reported by Mr. Howard Marsh. Yet, as I have already said, a neurotic history in the patient herself or in her family does not make her safe from lithiasis; indeed, some observers suggest that such persons are rather liable to this error of excretion. The following case is an instance of the insuperable difficulties of diagnosis which are met with:—Miss —— was seen by me and another medical man in the house of relatives. In herself and in the story of her folk the neurotic proclivity was very strongly marked; it was so strong as to dominate the interpretation of the instant symptoms, which I need not describe farther than to say that renal colic, whether neuralgic or calculous, formed a main feature of them, and that up to the time of which I speak several attacks of hæmaturia, of an unmistakable character and of no little severity, had occurred. Having seen many such cases, and more than one in which hæmaturia had occurred with mere nephralgia, I gave my opinion strongly that such and such only was the nature of the case before us. My medical friend agreed with me. However, such were her sufferings that an able and energetic surgeon who saw her afterwards at her own home urged still that stone was present, and thus her friends were induced to agree to an operation—the patient herself being willing to undergo anything for a chance of relief. And indeed even we were unable to deny that a stone might be present. The loin was therefore opened, and the stone was sought for with the ardour of a surgeon whose diagnosis was at stake; yet no stone was found. It is no strange thing to add that, at any rate during the interval which had elapsed between the operation and an accidental report to me, an interval of some weeks at least, there had been no return of pain: the operation of

nephrotomy, if unsuccessful as regards its primary purpose, is not infrequently the means in some obscure way of relieving the pain, at any rate for a time. So grievous is nephralgia, so futile, as a rule, are the means in the hands of the physician, and so safe is the operation nowadays, that an exploratory operation may be justifiable in doubtful cases even if there be no definite evidence of stone or gravel, and no hæmaturia; the more as indirect relief may accrue from the incision. Apart from the operation there is nothing to be done, in many cases, but to treat the general condition of the patient by improved nutrition, massage and the like, in order to enable the recreated system to throw off the burden: I say in many cases—for in some an aberrant function may be detected, and perhaps corrected with relief. Mr. Reginald Harrison has thrown some light on this dark subject by his observations on high renal tension, published in the article named in the list of references below. In some of the cases of nephralgia there is a high acidity of the urine; or indeed the deposition of gravel, however fine in grain or minute in quantity. Oxalates, again, may be the source of the irritation. Persons of a neurotic bent are victims to recurrent renal pains from such causes as these which, in ordinary persons, would escape notice or cause but a trifling discomfort. In *Visceral Neuroses* I report a case in which Dr. Solomon Smith, then of Halifax, repeatedly found a scanty fine gravelly deposit in the urine of a lady who had long been subject to this distressing malady of nephritic colic. It may be, indeed, that some aberrancy, perhaps of so trivial a degree as to escape recognition, lies at the bottom of all renal colic; on the other hand, the ordinary means of combating lithiasis and of rectifying the functions of the kidney too often fail to relieve the sufferers.

In concluding this section, I cannot altogether overlook the extraordinary statements of certain French physicians concerning the effects of hypnosis in cases of gastric and other neuralgias. M. Sollier (11), for example, declares that by inducing periods of hypnosis he can set up and dispel anorexia nervosa, and the rest of them at will—the patients bearing testimony to the favourable revolutions in their viscera. According to these accounts, maladies, like kings, can be made and unmade at pleasure; and by these proceedings the author declares himself able to carry out many researches on nervous dyspepsia and other stomach disorders. It is my duty to allude to these statements; but I think that it is no less my duty to content myself with an allusion to them until they are corroborated by other responsible observers.

T. CLIFFORD ALLBUTT.

REFERENCES

1. ALLBUTT, T. C. *Visceral Neuroses*, Lond. 1884.—2. GOODHART, J. F. *On Common Neuroses*, Lond. 1894.—3. HARRISON, REGINALD. "Painful Tension of Kidney relieved by Puncture," *Brit. Med. Journal*, 1896, vol. ii. p. 1126.—4. LEUBE. "Ueber nervöse Dyspepsie," *Deutsch Arch. f. klin. Med.* vol. xxiii. p. 98, 1879.—5. MARSH, HOWARD. *Clin. Soc. Trans.* vol. xxv. p. 195.—6. PARISER. *Centralblatt*

f. innere Med. 1896, p. 497.—7. PAWLOW, I. P. "Beiträge z. Phys. d. Absonderungen: Innervation d. Bauchspeicheldrüse," *Du Bois Arch.* 1893.—8. PAWLOW and SCHOUMOW-SIMANOWSKY. "Die Innervation d. Magendrüse beim Hunde," *Du Bois Arch.* 1893. —9. SOLLIER, PAUL. "De l'anorexie hystérique," *Revue de médecine*, 1891.—10. *Idem.* "Faits nouveaux relatifs à la nature de l'hystérie," *Il Policlinico*, 1894, Congrès de Rome.—11. *Idem.* "De la sensibilité de l'estomac, contribution à l'étude du dyspepsies nerveuses," *Revue de méd.* Jan. 1895.—12. STARLING, E. H. "Pawlow's Researches on Secretion," *Science Progress*, vol. iv. 1895-96.—13. WARDELL. Art. "Enteralgia," Reynolds' *System of Medicine.*

T. C. A.

DILATATION OF THE STOMACH

SYNONYM.—*Gastrectasis.*

Subject.—We are said to be in error if we make "a disease" of dilatation of the stomach. But there is no such thing as "a disease" apart from clinical convenience; and as chapters, and essays, and lectures are more and more devoted to this subject, I shall assume that the title is one which usefully indicates a fairly constant series of events. Under the name of dilatation of the stomach we do not include the capacious stomach of gross feeders and of those persons, labourers and others, who live not unhealthily on bulky food: by dilatation we mean a volume of the organ which is excessive in proportion to its ordinary work; and, again, after Rosenbach, we distinguish between relative and positive insufficiency of the stomach. We are told that certain tribes of men live upon an earth which abounds in organic matter; the normal stomachs of these men must be voluminous. Riegel in a careful paper seems to prove that a stomach, otherwise normal, may yet be of extraordinary capacity—a condition which has received such names as megastria or megalogastria. Trousseau reports 60-80 lbs. of food as the daily intake of a gross feeder. With chemical and motor functions absolutely normal, the stomach may descend to the navel or even a trifle beyond it; if the upper border be in place enteroptosis and "vertical stomach" are excluded from the diagnosis. Such colossal stomachs, however, must be of rare occurrence. Riegel's tests were inflation and soundings after digestion. Cottiers who live chiefly upon potatoes are said to possess stomachs capable of containing large quantities of this vegetable. In hot climates vast numbers of people live upon carbohydrates of low nutritive value—some of them, such as peas, being slow of digestion; but in those climates the quantities eaten at one time need not be very large; the quantity of fuel required for the bodily heat is not great, and inhabitants of the warmer climates are therefore small feeders.

If the work of the organ be persistently increased, and its nutrition be normal, the stomach, like the other hollow viscera, will strengthen itself accordingly—unless, indeed, the increase be too rapid, or overwhelmingly

large. If the work be increased—as, for instance, by a moderate degree of pyloric stenosis—but the bulk of the contents not increased, the volume of the organ may preserve its mean capacity: it may, indeed, be diminished; in rare cases it is enormously diminished, so as to present the form of the so-called "leather-bottle stomach" [*vide* art. "Cirrhosis of Stomach," p. 440].

Whatever its apparent size a stomach is morbidly dilated when food is delayed in it. Food may not stagnate in a big stomach; it may stagnate in a comparatively small one. The result of washing out six hours after a meal should be the basis of our diagnosis; not the

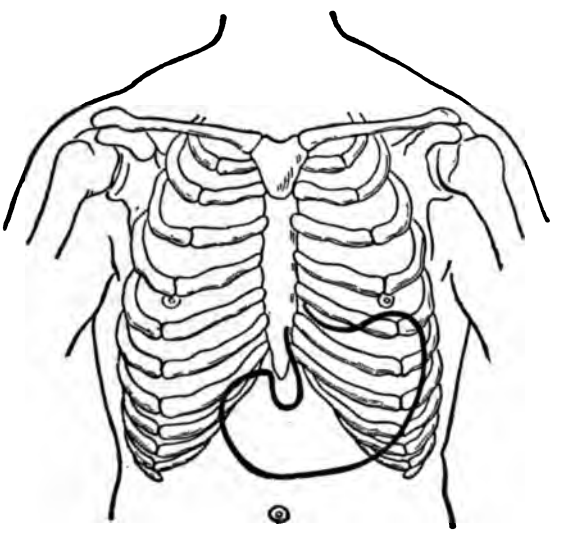


FIG. 19.—Normal position of the full stomach (after Luschka).[1]

measurement of the cubic capacity of the organ, which indeed during life is often impossible. And were it possible, there is no standard stomach for a given size of body; such an outline as Fig. 19 must be a mean.

The greatest measure of enlargement, apart from stenosis, may ensue when the work of the stomach and its contents are both increased; that is, when the food and its products are both bulky and indigestible. These two factors may fall together in very various proportions. In some cases the contents of the stomach, whatever their source, increase so rapidly, or the activity of the organ falls so fast, that work is put more or less in abeyance almost at once, and the distension cannot be compensated. These cases are called "Acute Dilatation." In other cases, where excessive demands upon the functions of the stomach come more gradually, the muscular coat may increase in greater or less degree with

[1] These diagrams appeared in the *American Journal of the Medical Sciences*, and were most kindly lent to the author by Dr. Pepper and Dr. Stengel (*vide* References).

the capacity; according to the circumstances of the particular case. Extreme instances may be taken from the big quasi-normal stomach of Riegel, and from cases of enlargement due to a narrowing of the pyloric channel (pyloric stenosis). If the pylorus be slowly constricted, the stomach may attain to a huge volume—to a capacity of seven or eight pints or more; but as it is unable duly to empty itself, and by the delay of its contents suffers injury both in motor and secretory power, it cannot retain its normal qualities as a merely big stomach may do. Why in some cases of over-work the stomach thickens rather than dilates is unknown. In the former case fibrosis may be the chief element, or very slow carcinomatous permeation; but, if writhings visible through

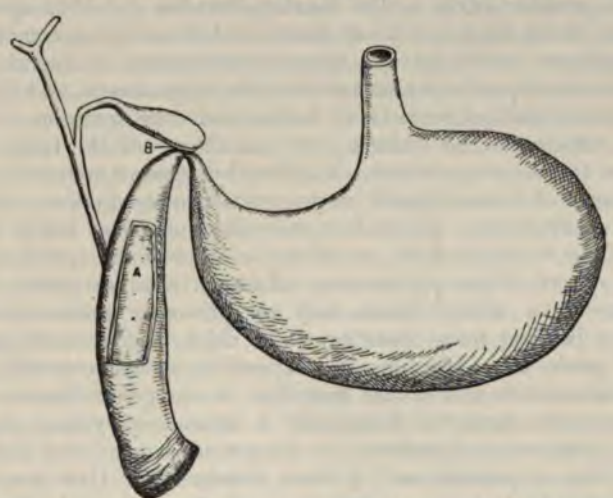


FIG. 20.—Diagram of stomach in which pyloric obstruction was produced by kink caused by adhesion to gall-bladder. (From Mr. F. Page of Newcastle-on-Tyne, published in the *British Medical Journal*, by permission.)

the integuments are to be taken as sufficient evidence of vigorous muscular friction, the "leather-bottle stomach" may be a very active one [*vide* p. 442].

Causation.—The causes of dilated stomach are—(A) Obstruction, usually about the pylorus; very rarely in the duodenum (4), or within the main cavity of the stomach itself. The pylorus may be thickened by disease benignant or malignant: it may be narrowed by the puckering of a cicatrix; by adhesions without, such as may arise on the irritation of gall-stones or other disease of neighbouring structures; by torsion due to dislocation, or by flexure of a deformed or dislocated stomach at its pyloric extremity.[1] (B) Enfeeblement of the function of the stomach, chiefly of its motor functions: (*a*) As a result of toxic causes, as in acute

[1] This cut is kindly lent to me by Mr. Page of Newcastle-on-Tyne and the editor of the *British Medical Journal* (*B.M.J.* 23rd January 1897). Mr. Page tells me that until he was able to inspect the parts he had hoped to perform pyloroplasty, which operation in two of his cases (out of four) had proved very successful.

rheumatism, pulmonary tuberculosis, septicæmia, and other infections. (β) Possibly by a sudden and overwhelming discharge of fluid into the cavity under some influence unknown to us; as in certain rare cases of a very acute and perilous kind described by Fagge and other authors under the head of acute dilatation. (γ) As a result of improper diet or drink—relative gastric insufficiency. (δ) As a result of some weakness in the organ itself either primary or due to some general failure of health—as in chlorosis, for example—whereby its chemical and motor functions, or both of them, are insufficient—positive gastric insufficiency. (ϵ) Possibly, but not very probably, as a result of an over-activity in the gland cells which secrete the hydrochloric acid of the gastric juice (hyperchlorhydria). Dr. Lauder Brunton (p. 392) speaks of a dilatation of the fundus of the stomach concurrent with spasmodic closure of the pylorus, which, he says, occurs in some cases of migraine. Such closure would depend upon irritation of the vagus nerve, and hyperchlorhydria also would not improbably concur with such irritation.

The causes of the sudden and instantly perilous form of acute dilatation (β) are very obscure. Rapid and dangerous as it is, the successful treatment of it may depend on prompt action based upon a difficult but imperative diagnosis. In its less overwhelming forms (α) it is usually the result of rheumatic fever, or of one or other of the specific infections such as enteric fever, pneumonia, influenza, or tuberculosis; and convalescence from acute disease may be grievously protracted thereby. Chronic dilatation from other causes is chiefly a disease of adults, and often of adults in middle life; if it appear in persons over fifty years of age its occurrence will arouse suspicion of malignant disease. Acuter dilatations are, however, frequently a disease of young persons, of children, and even of infants: it is not infrequent, for instance, in rickets; but, as pointed out by Mons. Comby, Prof. Hamilton, and myself, it occurs by no means exclusively in that malady. As Comby says, the pot-belly of rickety children is caused, in part at least, by dilatation of the bowels with undigested food, especially fæculent food. This is true, but I may demur that the external muscles of the abdomen are abnormally feeble in these children. Probably dilatation of the stomach, unless it be due to toxic causes, has no specific relation with any one form of lowered vitality. Whether in adults or children, the sequel of dilatation may come on quickly and be considerable in degree, and yet the ultimate prognosis may not be unfavourable: the dimensions, however, rarely or never attain those of the extremer degrees of mechanical dilatation. It must be remembered that the normal stomach has its times of distension, and even its occasions of immoderate dilatation; but the note of normality is that in such cases the organ has not lost the power of recovering itself. From the moment that distension is not followed by resilience morbid dilatation has begun; stress has led to strain, feebleness to perversion of function; food is delayed in the cavity, and the walls begin to suffer. Temporary dilatation, then, and even temporary insufficiency of the stomach, as of the heart, no doubt occur frequently in the run of life, and

in most cases without injury; but now and then, when stress is too great or too continuous or the organ is wanting in tone, the temporary becomes a permanent disorder. If again, as in Fagge's cases to be cited presently, the stress be sudden and excessive, the results may be instantly perilous and even fatal.

In obstructive disease of the pyloric end of the stomach, gradually established, no doubt a compensatory hypertrophy preserves the functional balance; but in time, as the distension surely increases, the compensation will fail.

On the other hand, "atony of the stomach" does not always, perhaps does not generally, end in definite and persistent dilatation. I have often been surprised to note an apparent caprice in this respect. Atonic and neurotic dyspeptics do not stand in great hazard of persistent dilatation. It may be that if the appetite be small the stomach is not loaded. The explanation, however, is not quite so easy. Some unknown factor, often perhaps of a toxic kind, connects dilatation with certain kinds of atony. If the vagus nerve be morbidly irritable, both chemical and motor functions must, for a while, be in excess; and both may fail as irritation ends in palsy of the nerve. The contents of the stomach, habitually small, may not suffer delay; at first, indeed, they may be hurried into the bowel with undue haste. Wilson Fox thought that dilatation of the stomach is apt to arise in hysteria and in hypochondriasis, and no doubt correctly; but the nervous disorder is as frequently the consequence as the cause of the dilated stomach, and, to speak generally, dilatation is the result rather of motor than of secretory failure.

No doubt a feeble stomach, although not increased in volume, and a dilated stomach, may stand in a like defective relation to an ordinary mass of food; still the volume of a hollow viscus is a very important factor in its work. Von Noorden, indeed, discusses under one head all cases in which the stomach does not duly pass on the food into the bowel, whether the state be one of atony or of stenosis. In all such cases dilatation is apt to occur: theoretically atony means dilatation.

The falling of the abdominal viscera, which is associated with the name of Glenard, will be described elsewhere [art. "Enteroptosis"]; but of gastroptosis, I may say that in moderate degrees it is not uncommon [*vide* p. 588]. In persons of steadfast nervous habit, and in such moderate degrees, its existence is usually harmless and unfelt; but in neurotic persons it may give rise to great uneasiness. Some displacement of the stomach is often apparent in cases of emphysema and of other deformity dislocating the inner organs of the body; and the degree of displacement of the stomach may be tested by insufflation. If it be considerable, pyloric kinking may occur with rapid aggravation of the state; and in any case much displacement seems likely to promote delay of ingesta, and thenceforward gastric insufficiency.

Duplay père, to whom the first systematic description of the symptoms of dilatation of the stomach is attributed by the French school, and who did indeed anticipate Kussmaul in many of the chief points of the

clinical description, divided the causes of the disorder into seven kinds: (i.) narrowing of the pylorus; (ii.) abnormal adhesions of the stomach; (iii.) destruction of the muscular fibres; (iv.) induration around the pylorus; (v.) atrophy of the muscular coat; (vi.) hydatid of the stomach; (vii.) palsy of the stomach. This division, I need not say, is both confused and imperfect. Our better knowledge of the subject and its treatment dates from Kussmaul's paper in the year 1869. In this interesting essay washing out with the pump was first recommended—a practice soon afterwards improved by the introduction of the tube and syphonage. In

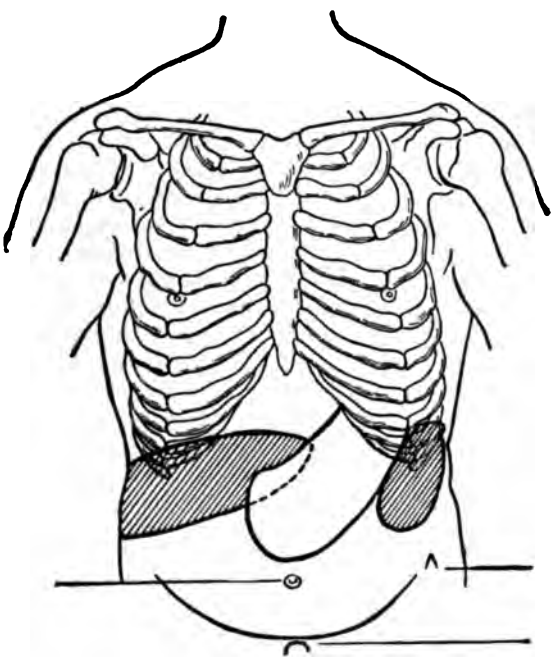


FIG. 21.—Enteroptosis, woman æt. 45; displacement of stomach, liver, and spleen (Pepper and Stengel)

1875 Penzoldt summed up the work on the subject which had followed Kussmaul's essay up to the time of his own, and his treatise also contains an admirable summary of the history of the disease. The number of publications on the subject since the date of Penzoldt's essay are innumerable, at any rate in this place. Penzoldt gives the honour of the clinical distinction between a tonic and stenotic gastrectasis to Johann Peter Frank.

From my own experience, I should find it difficult to say whether catarrh or primary atony of the stomach were the commoner cause of dilatation of the stomach not dependent on pyloric stenosis or constitutional disease. It is generally believed that catarrh of the organ is by far the commoner starting-point; but I am disposed to think that the frequency of dilatation of the stomach as a complication or sequel of acute

disease is not sufficiently appreciated in this estimate. It is easy during grave illness or during the term of convalescence to attribute debility and dyspepsia to "mere weakness": it is our business, however, to measure and define where we can, and I do not think that I have been unduly curious in detecting dilatation of the stomach in a very large number of patients, whether adults or children, in lingering and irregular convalescence after the infectious diseases, acute rheumatism, pneumonia, and the like. In these cases catarrh or imperfect secretion of gastric juice rather than atony of the pars muscularis may be the primary local term; and often no doubt catarrh and muscular atony are concerned together. However this may be, the fact remains that dilatation is often present in retarded convalescence; and that treatment of this sequel is necessary if a more rapid amendment is to be obtained. In any case atony of the stomach must lead to delay of its contents, to fermentation, and to secondary catarrh. In putting down dilatation as a disease of adult life —of persons of either sex between forty and fifty years of age—attention is too much fixed upon cases of local origin, and is averted from cases, such as those I have indicated, in which it is a secondary event. In cases due to more general causes the malady is quite as common in the young as in the elderly; indeed, as I have said, it may occur in children.

Bouchard suggests that in some persons there may be a specific and even a hereditary feebleness of the muscular fibre of the stomach, so that the organ does not contract vigorously between meals; thus the food tends to lie in it, and to offer a favourable soil for the microbes of decomposition —especially if, as he suggests, the hydrochloric acid be deficient. Whether then, in dilatation, the chronic catarrh be antecedent or consequent, the contents of the stomach are relatively or positively too much for it; and this state of things is the worse the weaker the muscular coat. On the other hand, let these causes be what they may—be they the gorging of food and liquids, wholesome or unwholesome, or lowered vitality due to previous illness or otherwise,—under any or all of these circumstances it is rare to find a dilatation equal to that of mechanical obstruction; in extreme cases of stenosis the stomach may reach the pubes, but never, I think, in atony. On the other hand, the physician who neglects the factor of dilatation because the stomach is not so blown out and washy as to force itself upon his notice, has an imperfect comprehension of his case, and an imperfect hold upon the means of cure; yet it is not a dozen years since a London physician of almost singular eminence said to me that of dilatation of the stomach, apart from pyloric obstruction, he knew nothing. "Nervous dyspepsia"—apart from the grosser dietetic errors, anæmia, the acute diseases, and the catarrh which plays in and out with these causes—is not, in my experience, a frequent or direct cause of dilatation. Of this, however, I shall speak again under diagnosis, and I have referred to it under "Neuroses of the Stomach." I repeat, however, that Dr. Lauder Brunton has found a transient condition of dilatation of the stomach, attended probably with contraction of the pylorus, in cases of sick headache.

Acute dilatation.—Dilatation of the stomach may occur not only in the overworked or irritated organ, such as we have had in our minds, or after a debauch (6), but the stomach, as I have said, may be suddenly overwhelmed and swift death may ensue. Two cases, originally published by Dr. Fagge under the title of "Acute Gastric Distension," are the classical cases of the kind. Both cases occurred in men, the one being thirty and the other twenty years of age. The symptoms and signs were much the same in both; the symptoms may be summed up as pain in the stomach, anuria and collapse; the signs were those of a largely distended stomach, more or less full of fluid. In the second case the stomach was emptied by the pump, but in spite of this measure death followed in a few hours, as it did also in the first case. I should add, however, that in the second case a sloughing abscess existed behind the duodenum. On section the mucous membrane was not examined in either case, but in both the stomach when relieved of its load returned to its normal dimensions. Dr. Fagge was disposed to attribute to the stomach itself that fluid which was found in quantity so enormous as to cause paralytic distension of the viscus. Sir William Broadbent lately told me of a case of dilated stomach under his care from which eight pints of fluid were removed by the syphon. No sooner, however, was this volume of fluid removed than the stomach began to refill and was rapidly distended again to its former dimensions. Happily such tremendous cases as these are very rare. Dr. Wilson Fox speaks of cases of sudden dilatation, and refers to Humby and Miller's cases which were marked by sudden invasion, vomiting and death in a few days. The causes of such cases as these are undiscovered; vomiting took place in most or all of them at the outset, but this palliative effort ceased as the distension became extreme: yet the stomach, on removal from the body, shrank back to something like its natural size, showing only white striæ, like lineæ gravidarum, upon its surface. Pepper and Stengel suggest that the immediate cause is spasm of the pylorus due to irritation by the contents of the stomach. Cases of an acute or sometimes of an overwhelming kind have also been reported by Andral, Peebles, Endmann, Kundrat, Oser, and Boas (6). Acute dilatation of the stomach, as ordinarily seen, is a much less destructive affair. Grievously as it may afflict and maim the patient, it is not usually so swift a messenger of death. In my own experience the malady has only occurred as the sequel or complication of other acute and debilitating diseases. In acute rheumatism, in the more active forms of pulmonary tuberculosis, in malignant endocarditis, in septicæmia, in influenza, or again in chronic non-febrile diseases such as diabetes, in all of which, as I have said, convalescence is often grievously delayed by a dilatation of the stomach, it either appears with some suddenness, or, if more gradual in its approach, is overlooked in the earlier stages which are concealed by the primary malady. In either case the dilatation may be well marked, but is never so great as in pyloric stenosis. There seems to be something more than mere atony about it; in acute rheumatism and pulmonary phthisis the toxic state of the blood, acting,

perhaps, upon the nerves (neuritis or myositis?), may intensify if it do not set up the mischief. Apart from pericarditis or endocarditis the heart may dilate in like manner. However this may be, the patient, bad enough to begin with, falls lower and lower; he wastes more rapidly, and his strength wanes, or fails to increase, more than the ordinary factors of the case quite account for. The physician who does not forget the liability of the stomach to fail in the fight, will examine the organ and readily discover the signs of no incipient palsy. In such cases recovery is still possible, but at heavy usury; the patient creeps out of his chamber wan, languid, and emaciated, and regains his health but imperfectly in a year or two. Not only is the general health slow in returning, but the stomach itself is almost the last part of the body to regain tone and functional vigour. Such a patient enters upon convalescence pined and reduced, and has to face the world again as a chronic dyspeptic. For many a month, or even for a year or two, a physical examination will demonstrate how hard it is for a hollow organ to recover a strain beyond the limits of its natural elasticity. In other cases I need not say that the dilatation of the stomach is but one more shaft from the quiver of Azrael; discovered or undiscovered, it is but a part of the company of death. A few months ago I saw a gentleman, advanced in years, in whom collapse had suddenly set in. He had made a very tedious but not unsubstantial amendment from pneumonia. The collapse was combated with some promise of success for some hours, but death was not averted. This calamity being unexpected and somewhat difficult to explain, a necropsy was made and dilatation of the stomach was found. Either the dilatation, which may have existed some time, had suddenly increased, or it had suddenly become too much for a weak heart. There was no gross disease of the stomach. Sir William Broadbent describes a similar case.

In very rare cases of acute dilatation, then, as in Fagge's reports, and in the case reported to me by Sir William Broadbent, the stomach, not incapable of contraction when relieved of its burden, seems to be flooded by an overwhelming weight of fluid, the origin of which is unknown; in other cases the dilatation, which may be compared to that of the heart, is due only to a relaxation of the tone of the musculature of the organ (δ); in others the nervo-muscular machinery seems to be poisoned by the toxins in the blood (α), as it is in diphtheria, enteric fever, influenza, and like maladies. In these latter cases the dilatation sets in with some acuteness, and is very obstinate—as incurable as a dilated heart or a dilated bladder. In chronic pulmonary tuberculosis the heart and stomach often present signs of dilatation and of poisoned function due to the absorption of ptomaines; but the dilatation is rarely excessive. Repair is possible in the more hopeful of these cases, but at best it is a slow business. On the whole, however, if we exclude the overwhelming attacks of Fagge, and those consequent on acute fevers, acute dilatation is probably a very rare disease.

Chronic dilatation, on the other hand, is common enough, and occurs

either as a primary malady, or as a secondary event in chronic exhausting diseases, such as phthisis, diabetes, and so forth. The muscular coat of the viscus, submitted to a strain positively or relatively too great, yields beyond its power of resilience, and remains in an abnormally extended state. In the acute form the primary fault, so far as the stomach is concerned, seems to be in muscle or nerve; in the chronic form the fault may lie in the muscular or secretory structure. The former is usually in fault when the stomach fails in the course of an exhausting disease, the latter when the malady arises in the stomach itself: in the former case, if the chemistry of the stomach be fairly maintained, dilatation will probably be averted or overcome. Bouchard (8) says that the stomach is dilated in 80 per cent of chlorotic women, but this is an exaggerated estimate. Dilatation of the stomach not infrequently arises from the ingestion of large quantities of fluid. I have often seen it in heavy drinkers of beer, and in persons who have abandoned themselves to the use of aerated waters or to the voluminous tea-drinking not uncommon in the more temperate class of miners, engine-drivers, stokers and like dusty, thirsty labourers. Unfortunately these workers also consume with their tea a large quantity of carbohydrates; they leave home in the morning after a breakfast of hot tea, bread and butter and cakes, and carry with them more bread and butter and a can of tea in their pouches; and on this they live until the return home, which, in the case of railway men, may be at a very uncertain hour. Thus the stomach, wind-blown rather than hard-worked, does not hypertrophy after the fashion of the stomach of the over-feeder, whose table is well spread, and whose diet is as varied as his digestion is vigorous and his food nutritious, but tends rather to dilatation. The big, well-fed stomachs are soon reduced by proper treatment when dyspepsia drives their owners to the doctor; but the thin, inflated, and ill-nourished stomachs do not contain within themselves the same means of repair. Moreover, the stomach of the glutton often makes more than ordinary quantities of gastric juice; but the distension of the walls of the ill-fed stomach, thinning out the glandular structures, impoverishes them; and the food finds, not an increased measure of solvent, but a positive deficiency. Thus digestion is delayed, flatulency increases more and more, the stomach gives way more and more, and the evil grows in geometrical ratio. A large bulk of fluid, then, tends to dilate the stomach: if the food be generous the dilatation is well compensated by hypertrophy, and the organ can pull itself together; but if the food be poor and windy the strain acts upon an organ the natural elasticity of which is already impaired: in such cases, as I have often observed in miners, the results may be irremediable.

There are two kinds of acid dyspepsia: the one, which is of rare occurrence, depends upon an excessive secretion of hydrochloric acid; the other upon the generation of lactic and butyric acid by the activity of the several bacteria of these fermentations. With the former kind we have now little concern: it is said that hyperchlorhydria is a cause of dilatation, but on slender grounds: the latter kind is the result of

a deficiency of secretory or of muscular power, or of both, so that the contents of the sac fall more readily to those microbes which would be hindered by the mineral acidity of the vigorous stomach, and, being delayed in their progress through the pylorus, give easier occasion to the agents of fermentation. Thus initial feebleness of gastric juice leads to delay of the chyme; the chyme thus delayed and imperfectly reduced falls the more readily into fermentation; and the products of fermentation distending walls already lacking in tone handicaps them more and more in the race of peristalsis. The stomach is thus in a vicious circle; its very feebleness increases its burden, and dilatation seems inevitable. If in addition to these conditions an especially flatulent food—such as tea and bread and butter—be consumed in large quantities, things go from worse to worse; the stomach, like a dilated heart, is stretched beyond recovery, and, as Cohnheim says, the patient has now a vinegar, now a gas factory in his body. There is no superstition more tenacious of life than that which prescribes carbohydrates to all dyspeptics as "so digestible"; and into weak stomachs ready to dilate is thrown a mass of such a dish as rice pudding,—a bulky food, imperfectly salivated and peculiarly apt to fall into decomposition with the disengagement of volumes of carbonic acid gas.

A woman of about fifty once came under my care for "Heart Disease." I found the rhythm of the organ as irregular as it well could be. The stomach was dilated. She had been nursed and kept almost entirely in bed for three months; without altering the other conditions in any way we took her off the invalid diet of pap and tea and the like, put her upon a drier diet of tender meats with but little starchy matter or cellulose, and cut off tea and coffee; in a fortnight she felt a different person and her heart had become nearly regular, as regular as after so long a bad habit it was likely to become. She soon got so far well that I lost sight of her. No doubt the heart was dilated, but the flatulent and distended stomach was a great aggravation of her disorder.

A dilated and flatulent stomach may disturb and gravely disturb a normal heart: when the heart like the stomach is weak and dilated also, the disturbance is at once more distressing, more persistent, and more mischievous. If one of the results be a rise of venous pressure in the abdomen, the stomach suffers more and more, yields more and more, and thus the embarrassment of the heart is multiplied. Dilatation of the stomach does not attain any very great dimensions in heart disease, but is a frequent and very troublesome addition to the disorders more directly cardiac.

Spasmodic asthma is, of course, grievously aggravated even by moderate gastrectasis; and it is said that this extension may set up asthma even in persons to whom it would otherwise have been unknown.

Loss of tone in the muscular fibre, then, and a defective gastric juice, conditions common enough in chronic diseases, are fertile sources of dilatation of the stomach—a condition much aggravated if the so-called "dyspeptic" is put on carbohydrates with the notion that they are digest-

ible. The next factor to command our attention is gastric catarrh. The state I have just indicated is one always ready to call forth a catarrh; on the other hand, a gastric catarrh, by reducing the value of the gastric secretion and promoting fermentation, is a potent means of setting up dilatation. Gastric catarrh, again, must not be treated, as so commonly it is treated, by carbohydrate food. The contents of the catarrhal stomach tend too much towards alkalinity, and thus to the harbourage and cultivation of bacteria; it should be our business to make these contents acid with a mineral acid, and to give finely-minced, tender meats with hydrochloric acid after the meals. As Dr. Sidney Martin has well insisted, pepsine may

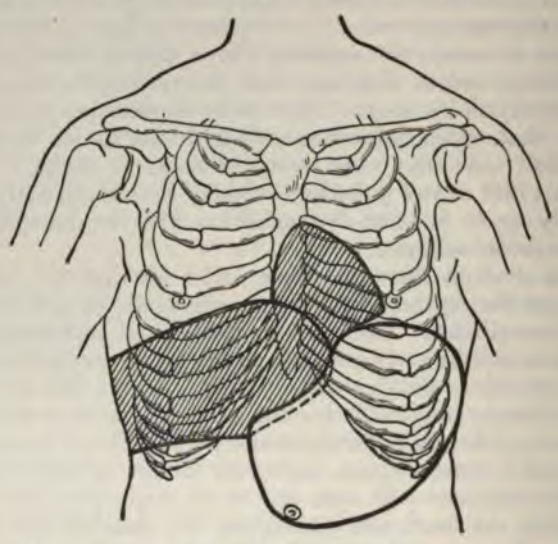


FIG. 22.—Outline of stomach measuring 17·5 cm. vertical, and 22 cm. transverse. From case of a man æt. 50. Atonic and obstructive dilatation, due to a cicatrix in the anterior wall and adhesions surrounding the pylorus. (Pepper and Stengel.)

be needed, hydrochloric acid is certainly needed. Pepsine is a more persistent element in the secretion, and, moreover, as a ferment its work is not, like the work of hydrochloric acid, in direct proportion to the quantity present. Dr. Saundby, in his article on diabetes in this volume, says that the dilated stomach of diabetes is catarrhal and the mucous membrane often thickened; and Dr. Soltau Fenwick (21) seems to hold the same opinion of its origin in phthisis. Dr. Fenwick says that in phthisis of moderate severity some dilatation is almost always present; and in later stages, if the case be slow and protracted, the greater curvature may fall several inches below the navel, and even to the pubes.

Dilatation, then, is variously associated with catarrh, as cause, concomitant, or consequence. The cavity is never thoroughly emptied, and thus always contains a remnant of decomposing food to taint all that comes in. But this retention is at its highest in cases in which there

is some mechanical obstacle at the pylorus. It is in these cases, whether of cancer of the pylorus, of fibrous thickening of the part, of external adhesions, of torsion, of cicatricial closure due to past ulceration or possibly to corrosive poison, that dilatation reaches its highest expression. It is in cases of pyloric obstruction that the stomach attains those portentous proportions which enable the patient to deliver himself of vomitings which fill a bucket. Curiously enough, in pyloric obstruction the stomach, instead of yielding, sometimes contracts, its coats becoming enormously thickened and its cavity reduced. In a man aged about forty, whom Sir W. Gull saw with me at the close of his life, and in whom the symptoms pointed to a greatly thickened and contracted stomach, this state and pyloric stenosis were found after death. The stenosis was due to the scars of three or four simple ulcers. It is remarkable that some three years before his death the stomach was considerably dilated, and that this dilatation gradually gave place to the contraction. The walls were of extraordinary thickness and the cavity very small. There was no carcinomatous infiltration. The thickening was largely cirrhotic, yet during life peristalsis was very active and visible [*vide* p. 443, the same case].

Dilatation of the stomach is apt to follow surgical operations upon the abdomen, and to give rise to alarming symptoms which may be much relieved by lavage.

By *torsion* we mean that a distended and overloaded stomach, dragging at its pyloric attachments, causes acute flexure there whereby the issue of its contents in the normal direction is still further impeded. Sir William Broadbent has added to this certain cause of increasing gastric dilatation the supposition that in some persons the lesser omentum is too short and the suspension of the pylorus unduly high. In such cases the kink at the pylorus would form more readily, and such persons would have a predisposition to dilatation of the stomach. For information concerning the mechanism of dilatation of the stomach by displacement of the kidney forward so as to press upon the fixed descending portion of the duodenum, and to obstruct the passage of the chyme, the reader is referred to the article on "Nephroptosis" in the next volume of this *System*.

Symptoms.—It must be remembered that in not a few cases, more especially in those in which gastrectasis is a complication or sequel of some other disease, the patient may not complain of any characteristic symptoms; in the midst of his other troubles some dyspepsia, flatulence, or gastric discomfort may pass unnoticed, or be accepted as part and parcel of the constitutional state. In all cases, therefore, in which dilatation may be suspected, a careful physical examination must be made. The symptoms of chronic dilatation of the stomach are often of a general kind only, such as feebleness, anæmia, emaciation, thirst, scanty urine often abnormal in its constituents, heavy aching limbs, sallow and hollow-eyed face, pale flabby tongue, sour breath, dryness of the skin and other tissues, chilliness even to ashiness or cyanosis or subnormal bodily temperature, vomiting, and certain nervous symptoms, sometimes of a peculiar

kind and due more or less perhaps to peculiar causes, as will appear hereafter. Pain and vomiting may also be present and more definitely indicate the nature of the disease.

Pain.—The pain in dilatation is not the ordinary pain of dyspepsia, or is that pain with a difference. It is not felt especially after food; it is not associated with eructations or acid risings; it is not periodic or even very variable.[1] It is not the acute pain of gastralgia or of some cases of malignant disease, it is rather a sense of weight or oppression; but it is none the less wearing and intolerable. This pain is due to the burden within the stomach, and is quickly relieved for a time when by vomiting or syphonage the burden is removed. It is characteristic rather of dilatation mechanically produced.

Thirst is not less distressing than pain. Full of slop as he is, the patient burns with thirst, as absorption from the anæmic and perhaps catarrhal walls of the stomach is almost at a standstill, and the liquids drunk by the thirsty sufferer lie in the sac stagnant until rejected; the thirst which follows even ordinary and occasional vomiting is well known to every reader of these pages.

The urine for the same reason is scanty and usually deficient in chlorides. It is often alkaline and contains triple phosphates. Dr. Sidney Martin tells us that it may contain excess of ethereal sulphates from the putrefactive changes; though such changes (as he observes) are more usual in the intestines than in the stomach. Albumin is not an uncommon impurity of the urine in cases of gastrectasis of the extremer degrees; or at an earlier stage albumosuria (peptonuria) may occur, as in carcinoma or ulcer of the organ. Acetonuria is occasionally seen in cases of dilatation of the stomach; for a further account of this symptom the reader is referred to the article on "Ulcer of the Stomach" (p. 532).

The Bowels.—Dryness of the skin and indeed of all the tissues, and constipation of a most obstinate kind, are conspicuous—the fæcal masses being shrunken and hard. Conversely, these symptoms should lead to suspicion of dilatation or other atrophy of the stomach. It is said that diarrhœa occurs in some cases of gastrectasis. I am again indebted to Sir William Broadbent for the account of a lady once under his care for gastrectasis, in whom the stomach would empty itself from time to

[1] A stomach which is capable of frequent upward discharges of its contents, whether solid or gaseous, is not seriously dilated. I omit all allusion in the text to those cases in which sulphuretted hydrogen is thus discharged: the symptom is a very noisome one. The gas is antagonistic to the lactic acid fermentation; it is often associated with hydrogen and marsh gas, and Boas says it is a result of dilatation (6*a*). The symptom is of rare occurrence and difficult to explain; but in my experience it is not characteristic of dilatation. It may be due to abnormal patency of the pylorus, so that intestinal gases rise into the stomach. I recently saw a bad case of the kind with Mr. Copley of Wisbech in a neurotic young woman. The stomach was not permanently dilated, and Mr. Copley, by test lavage, believed it occurred independently of albuminous food. Indeed, the phenomenon would recur after the stomach had just been washed out and was presumably empty. Indol has been detected in the stomach in rare cases, and sulphuretted hydrogen, like indol, may be due to the presence of the colon bacillus (46*a*). Hoppe-Seyler (27) analysed the gases in eleven cases of gastrectasis, and found varying proportions of hydrogen, nitrogen, carbonic acid, and oxygen. The gas was usually inflammable.

time with a rush from the bowels. The recumbent posture seemed to be the means of enabling the viscus thus to unload itself; after retiring to rest the patient would have a sense of the gushing of fluid within her, and on seeking the closet a profuse liquid discharge would issue from the rectum. After such a discharge the stomach was no longer perceptible by the physical signs which had previously made it very manifest. Ultimately an autopsy was obtained and non-malignant thickening of the pylorus without stenosis was found. In the upright position the pylorus was probably closed by acute flexion of the part at the point of suspension.

Emaciation.—The late Dr. Sutton tells a horrid story of an autopsy on a case of his own in which he found food impacted layer upon layer in the stomach and gradually so consolidated there that the cavity was represented only by a small passage, in the middle of the mass, through which fluids had trickled into the duodenum. This may serve as an extreme instance of the cessation of all nutritive function in the organ; and if the state of the ingesta is usually rather that of a sour fermented "swill," the absorption of nutritive matters is none the less in abeyance, and none the less is the patient's body reduced by a dry atrophy. Moreover, such food as may be absorbed is by the abnormal decomposition reduced to almost worthless elements. The appetite is bad or capricious.

Temperature.—As in other cases of reduced vitality the temperature often falls below the normal range. From lack of fuel the warmth of the body is spent and not replenished. The patient feels chilly, is cold to the touch, pinched in face, and may become even ashy or cyanotic. The pulse is often very slow, it is always feeble. Such changes may be due in part to auto-intoxication.

Vomiting is not a constant symptom. The small part taken by the stomach itself in the act of vomiting is probably less and less possible as the stomach is extended; the reflexes arising from its interior surface are weakened, and until the irritation becomes extreme the vomiting machinery is not called into action. Speaking generally, vomiting is more frequent and characteristic in cases in which dilatation has come on gradually from mechanical causes, in the midst of comparative health, than in cases in which the viscus has yielded under the shadow of exhausting constitutional maladies. The vomiting, when present, is not frequent, it does not return after most of the meals, as in cancer without dilatation, but three or four meals, or the meals of three or four days, accumulate. Moreover, individuals differ a good deal in readiness to vomit. For like reasons eructations of food are unusual; the stomach, now unable to expel small quantities from time to time, when its burden becomes intolerable, is passively emptied by a sudden impulse of the abdominal machinery. The dyspeptic who suffers from acid eructations probably has no dilatation of his stomach. Thus, as I have already warned the reader, the dilatation is often latent. Unaccountable emaciation, obstinate constipation, scanty urine, and failure of bodily heat should move the physician, therefore, to examine the stomach care

fully; and this the more as dilatation without vomiting may signify an acute and very perilous condition. Whatever the history of the disease, vomiting is a conservative process; by thus unloading himself the patient gets along. In a case of large ectasis of gradual initiation the act tells its own tale. After a time of increasing repletion, whether by food and drink, or by some such weeping of the walls into the gastric cavity as Fagge supposed and Broadbent observed, the burden becomes intolerable. Under the weary education of affliction the abdominal muscles become trained to the new labour, and at a certain height or at a certain weight of gastric contents vomiting begins in earnest. The nausea described by most authors may sometimes be felt, but in my own cases nausea has not been usual; somewhat suddenly the contents of the sac are cast up by contractions of the abdominal muscles so violent as to shoot the foul stream from the mouth as from a pump. As if by repeated strokes of a piston, the spouting fills the pail until the onlooker wonders whence it all can come! The vomit, if held up to transmitted light, is usually darkish gray in colour, and streaks of a lighter gray mucus are seen to be suspended in it; on the top is a darker, brownish froth: at the bottom is a deposit of solid matters; chiefly food-remnants mixed with mucus, sarcinæ, yeast cells, and other microbes. By the microscope or with the naked eye matters swallowed many days or weeks previously are often to be found. Sir William Broadbent found (private report to myself), on a certain 31st of March, in the washings of the stomach of a woman suffering severely from gastrectasis a remnant of preserved ginger, which had been eaten on the previous Christmas Day. She was certain that she had never eaten of ginger since that day. The stuff smells of the swill tub, and is mostly acid with the acids of fermentation, though in some cases hydrochloric acid is to be detected; often indeed in no small quantity (Cahn and v. Mering): it seems, therefore, that hydrochloric acid, even in normal quantities, does not always arrest fermentation. I suspect that in the atonic cases free hydrochloric acid is always in defect, although it may be abundant in the hypertrophied stomach of stenosis. The vomit probably contains also certain poisonous products of which little is accurately known, but which may be charged with peril to the patient nevertheless.

Unfortunately the vomiting, violent as it may be, does not quite empty the cavity; and, as in the case of other hollow organs, a little puddle remains to contaminate all food subsequently taken in. The relief, however, such as it is, is welcome to the patient; for some hours to come he is free from his distress.

Kussmaul, under the name of "Tiefstand," describes stomachs which by means of tight lacing, or other dislocating agency, are thrown into a loop so that the pylorus approaches the cardia. In this change the smaller curvature becomes acute, the large curvature extends, and the whole organ falls deeply down into the abdomen. In these cases, he says, vomiting becomes difficult or impossible; so that in them syphonage is of urgent importance.

Nervous symptoms.—Besides the depression of spirits which cannot fail to be present in an abdominal malady so persistent and so full of misery, there are, in not a few of these patients, symptoms which suggest a more specific source. The weary and disheartened sufferer falls into a melancholy; he is annoyed by strange sensations; he is fretful, sleepless, and subject to night-mares; his head aches, and with the headache may be tinnitus, vertigo, or visual disturbances; he then may fall into a syncope, or, worse than this, tetany or convulsions may appear, or even a coma, in which the patient may die [*vide* art. "Tetany," in a later volume of this *System*]. These alarming and perilous events seem to depend upon the formation of poisons in the stomach; Ewald, indeed, in a case of tetany of intestinal origin discovered a kind of ptomaine in the urine. Trousseau's observations on tetany in the diarrhœa of children are well known. Bouveret and Dévic injected fluid from the stomachs of such cases into animals and thereby set up tetanic convulsions. The same authors state that even in cases of such severity an excess of hydrochloric acid will altogether prevent the formation of these poisons. Alcohol, they say on the other hand, favours their formation; and till this observation is refuted it will be our duty to avoid alcohol, if possible, in our dietary or treatment. In discussing the causes of this poisoning we are reminded forcibly of Bunge's view of the stomach, not as the chief organ of nutrition, a function which he attributes rather to the intestine, but as the seat of the protective function of the disinfection of ingesta before they go farther. How important the stomach chemistry is in the aversion of such dangerous poisons as the typhoid and the choleraic is well known. It may be, therefore, that the brew of poison is due negatively to gastric inefficiency rather than to positive concoction. In the Czerny-Kaiser experiments, when the stomach was removed from dogs, the animals were poisoned by foul flesh which in the normal state they would have devoured with impunity; and Pawlow observed a like result in dogs after section of the vagi [see art. "Neuroses of the Stomach," p. 464].

Alcohol, again, as Tappeiner has indicated, may be mischievous indirectly by facilitating absorption. Bamberger, although believing that auto-intoxication is at the bottom of these accidents, reviews among other possible causes a reflex irritation arising from the walls of the stomach, and again the desiccation of the tissues. The most important "desiccation," surely, must be the thickening of the blood from imperfect absorption, which I have compared to that in cholera; and, as in this disease, it is apt to produce morbid phenomena of its own, possibly even the tetany; certainly cramp is common in gastrectasis. At present the prevalent working hypothesis is that one or more soluble poisons, formed, or at any rate present unneutralised in the stomach, are the cause of tetany and other such accidents. Acute nervous disturbance and death have occurred not a few times during lavage; and, beneficent as lavage is on the whole, it is alleged that tetany and worse than tetany have been disasters due to this operation. Dr. Soltau Fenwick

has carefully considered this matter. That lavage creates the dangerous factor seems improbable, but it may determine the absorption of it. Dr. Dreschfeld once saw tetany appear spontaneously without lavage in a case of dilated stomach [*vide* art. "Ulcer of Stomach," p. 533]. Bouchard, amid much that is fanciful, has drawn our attention forcibly to auto-intoxication, and we cannot ignore his arguments, even if we think some of them exaggerated; other physicians also have insisted upon some such explanation of phenomena, which, obscure as their origin may be, are none the less hazardous and even lethal. Senator was, perhaps, the first to suggest the possibility of this kind of auto-intoxication. Yet, although in the face of such accidents as slow pulse, tetany, convulsions, coma, and so forth, we cannot but suspect self-poisoning, Müller sought for ptomaines in the dilated stomach of such a case with negative results; and although Brieger's peptotoxin can be obtained in the laboratory from fibrin, it has not been found in the stomach. Lorenz, von Jaksch, and von Noorden have found acetone in the stomachs of persons apparently poisoned by their own organs; acetone might arise out of milk fermentation, but as it is more or less oxidised on absorption it could scarcely accumulate in dangerous quantities. No great harm can come of the decomposition of fat or of carbohydrates; we have to look to albuminous ingesta to generate toxins, but conversion of these into toxins must be checked in the presence of a large quantity of acid, even if the acid be organic; and as a matter of fact, such products of albuminous decomposition as phenol and indol are very rarely seen. However, from this point of view it may be unwise to cut down the amount of carbohydrate food too much. Obscure as the whole subject still remains, these theoretical considerations must not be ignored. Whatever the mode of generation of toxins, if such there be, careful washing should rather cleanse and prevent evil than be itself the cause of the tetany or convulsions, as has been alleged against this mode of treatment; though it is conceivable that washing may favour absorption at some critical moment. These dangerous accidents at most are very rare; I have been washing out stomachs for fifteen years, at one time on a large scale; yet I have never seen tetany or convulsions in such cases, either during syphonage or otherwise.

Finally, Bouchard describes an arthritis in patients suffering from dilatation of the stomach, and attributes it to the absorption of poison from the alimentary canal: I cannot call to mind any instance of this association of diseases.

Physical Signs.—*Inspection.*—Emaciation is so commonly associated with gastrectasis that inspection of the abdomen is often useful. Not infrequently, indeed, the outline of the distended organ can be seen, and demonstrated to a class; and the breach thus made in the symmetry of the contours of the abdomen is very characteristic, the fulness lying towards the left hypochondrium. The abdomen need not be protuberant as a whole, and usually is not. To see the upper border the light should be at the foot of the bed; to see the lower border, on the other

hand, the light should come from the head. When the main contours of the stomach are visible the organ is temporarily or permanently deranged; but it may not be dilated; on the contrary, it may be contracted and thickened. If the lower curvature do not transgress the parallel of the navel the distension may be within the bounds of the normal, or at any rate the stomach may be able to recover itself in due course; but in such cases the upper contour will probably be invisible. In many thin persons after a meal the flatulent stomach may be recognised as a protuberance in the epigastrium; but after a short time it recedes within its proper limits.

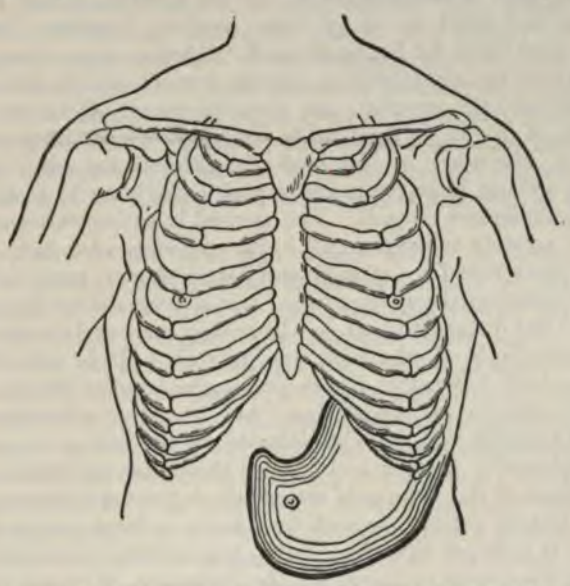


FIG. 23.—Obstructive dilatation—non-malignant stenosis of pylorus; man æt. 35 (Pepper and Stengel).

Sometimes the diagnosis is facilitated by making the patient stand up and placing him so that the light falls obliquely upon the abdomen. Any variation between the vertical and horizontal postures will of course be noted.

If, however, the smaller curvature be visible, and the larger curvature sweep down to the parallel of the navel, or one to four finger-breadths below it, rising again into the left hypochondrium up to the tenth rib, there is more than a transient distension; the stomach is probably stretched beyond its capacity of resilience. In gross feeders the abdominal walls are usually too fat to permit the outlines even of a big stomach to be seen. In rare cases the lower margin may sweep down even so far as the pubes: it may drag the pylorus from under the liver downwards, so that this structure, even when free from disease, may be perceptible to the touch; if it be thickened it can be grasped easily enough, or adhesions of the pyloric portion to neighbouring

structures may be detected. Here again, however, the possibility of a mere displacement without great dilatation must not be forgotten. The position of the upper contour and the other facts of the case will probably prevent any error in this respect. In displacement the prominence of the stomach below its normal position contrasts with the epigastrium left hollow by its descent. The upper border often travels up and down with the breathing, or seems to do so.

If the outline of the stomach be not visible, or be indistinct, or if its volume be not sufficiently manifest, further means must be employed. Leube introduced a stomach-sound for the purpose, which I need not describe, as it proved to be of little service; I believe, indeed, that it is no longer used by Leube himself. Another way of mapping out the stomach is by administering to the patient an alkaline carbonate followed by an acid, so that gas may be given off in the stomach, the volume of which is thus made manifest. Von Ziemssen prefers this plan to any other of the kind; he gives to an adult man seven grammes of sodium bicarbonate and six of tartaric acid; to a woman he gives one gramme less of each. The method is an uncomfortable one for the patient, possibly injurious, and in my experience has been difficult to control. I have found it a simpler and better plan to pump air into the stomach by means of the syphon-tube; this is easily done with an inflating syringe, and the quantity of air can be readily increased or diminished to the advantage both of physician and patient. It is said that if the pylorus be slack distension of the duodenum can be detected; this I have never seen to my knowledge; but in atonic dilatation without stenosis the inflation test may be defeated by the escape of some of the air into the bowels. Pepper and Stengel think that by inflating quickly at first a spasm of the pylorus is set up which prevents this escape.

To try to map out the stomach by pouring a large quantity of some fluid into it is to forget that the stomach is movable as well as distensible. Manometers have been contrived for the estimation of elastic pressure of the walls of the viscus, but they are idle toys.

Peristaltic movements of the stomach are often to be seen; and if they do not occur during quiet inspection they may be elicited. Flicking with the wet end of a towel is said to be a good way of producing them; but if they are to be got, manipulation will suffice to bring them out. If the left flank and epigastric region be kneaded, rolled, or chafed, the movements come out, if the stomach be capable of them; a tumour the size of an orange may rise in the cardiac portion and slowly and sinuously travel towards the pylorus; this movement may be repeated either spontaneously or by a renewal of the handling. Reverse peristalsis is occasionally seen, and is significant of pyloric obstruction. In most cases, however, the sinuous movements of the walls are not fully visible as such; but from a flat epigastrium a large boss or phantom tumour may suddenly form under the eye, and falling again may reappear at another part of the gastric area, the intermediate progression, if such it be, being lost to sight. A soft and flat epigastrium

may thus at intervals "rise up like a quartern loaf." Sometimes hand-pressure upon any tumidity about the pylorus will force up the fundus into a boss. Movements of this kind are readily apparent only in cases of a chronic course, in which the coats of the stomach are hypertrophied, or at any rate active; in cases, that is, of pyloric obstruction of whatever kind, whether the stomach be dilated or contracted: on the other hand, it is probable that palsy of the gastric walls is never complete except in intense cases.

For inspection of the patient in the erect position when gastroptosis is suspected, the reader is referred to the article on "Enteroptosis" (p. 587).

Palpation.—Sometimes the hand passed very lightly over the abdomen will detect a prominence of the stomach when the eye fails to do so. In the larger number of such cases the meaning of the prominence is scarcely to be mistaken; the organ gives to the hand a sensation as of an air-cushion, which, for my part, I have often found very characteristic. If peristalsis arise under the hand at the same time the diagnosis is easy; but the walls may be too slack or the contents not such as to produce this action.

The "splash" is the next sign which we try to obtain. This is sought for by placing the right hand under the patient's left flank, and the left hand upon the navel; the two hands are then sharply thrust to and fro, either alternately or together; or the two hands may be placed on the abdomen, and the sudden displacements repeated, or again by grasping the iliac crests the abdomen may be shaken as a whole. A little patience may be required as, even in a dilated stomach, a definite splash may not be obtained at first; it would seem that some peculiar disturbance of the gaseous and fluid contents is necessary to produce it: the splash once obtained, however, may be reproduced at will for some time. I am disposed to say, in spite of contrary opinions, that if a definite splash of a well-marked character is present, dilatation of the stomach or colon certainly exists. The difficulty is that squelchy sounds may be produced which may indicate dilatation, but are not conclusive. Practice alone will enable the physician to distinguish between the mimic squelches and the unmistakable splash. The former may be obtained in stomachs or colons of normal dimensions; and they are not uncommon in women, or in spare, dyspeptic, nervous persons of either sex, who are also subject to the so-called crying bowels—spontaneous and long-continued gurglings which are sometimes so annoying as to drive the sufferers out of society. Some years ago I pointed out that in some neurotic girls this noise is rhythmical with the respiration, or is called forth by deep inspiration. In these persons the respiration, even when forced, is almost wholly diaphragmatic, and the troublesome disorder may be removed by promoting the expansion of the chest. In patients such as these manipulation often brings out squelchy sounds which simulate the splashing of gastrectasis, but they are not identical with it. Still, between such sounds, and those which may indicate moderate degrees of dilatation, doubt may readily arise, or distinction be impossible. The difficulty

is the greater as some degree of gastric dilatation is not uncommon in chlorotic girls, and impedes their recovery. False splashing is often produced in the colon, or possibly in the small intestine. In such doubtful cases the splash should be sought before food is taken, as for instance in the early morning after the patient has drunk a glass or two of water. Flaccidity of the muscular wall of the abdomen greatly facilitates the examination. I am rather indisposed to admit that true splashing is found in a stomach which is but temporarily distended, say by a heavy and flatulent meal. The fluid gets away pretty readily and the walls contract upon their contents. If the stomach be full, that is to say, adapted to its contents, splashing cannot well be obtained.

In not a few cases the stomach may be found pressing up the diaphragm and rising into the region of the left thorax. In such cases the heart sounds may have a consonating or metallic quality. I have not been able to satisfy myself that these signs are indicative of dilatation of the stomach, of more, that is, than a temporary windiness. On the other hand, we hear of cases in which the liver, displaced by the stomach, or swollen by the task of converting the poisons generated in the stomach, and in some persons thrust down by belts or lacing, falls downwards and drags, so it is said, the kidney with it. No instance of this series has come under my notice; and indeed I hesitate to believe that gastrectasis initiates such changes, though it may accompany them.

Percussion. — This means is less valuable in detecting gastrectasis than might be expected. For me at any rate there is often a great difficulty, especially in the obscurer cases, in distinguishing by percussion alone between resonances of stomach, of colon, or even of distended small intestine. The colon, indeed, may override the stomach. When used after inflation percussion is of some help; the upper border especially may be defined, and from this some estimate may be made of retained ingesta and secretions: in a few cases a shifting line of dulness, as of shifting contents, can be found when the patient changes from the standing to the supine position. Whether over stomach or bowel the percussion note may quickly alter as the cavity changes its form. The spleen may encroach upon the area of the fundus, as in fevers; or a large liver may be mapped out; but little is to be learned from percussion of the stomach which is not to be better learned from inspection and manipulation. Osler states, on Pacanowski's authority, that the greatest vertical diameter of gastric resonance in the normal stomach varies from 10 to 14 cm. in men, and is about 10 cm. in women; but, as I have said, to measure the capacity of stomachs in general by so many centimetres of resonant area on the surface of the abdomen cannot satisfy a cautious observer. There is no stomach note properly so called; with the tone and tension of its walls the resonance varies through a wide compass.

Auscultation is of very little use, whether practised in combination with percussion, as recommended by some authors, or alone. Combined auscultation and percussion may interest the observer in a case in which

the diagnosis has been made by other means, but as a foundation for diagnosis auscultation is of little service.

Morbid anatomy.—It is rather difficult to deal with this part of the subject without travelling out of it. With the various causes of mechanical dilatation — whether cicatricial, fibrotic or carcinomatous, or due to pressure, or adhesions of external origin, and so forth—I have not to deal; these changes are described elsewhere. In like manner, catarrh of the stomach and atrophy of the organ, although often associated with dilatation, are not an essential part of it, and are likewise described elsewhere.

Of the dislocations of the viscus I have spoken incidentally; it will suffice here to say that, so long as the attachments of the end of the sac hold, the lesser curvature is bent more acutely; but the stomach does not fall as a whole. When, on the other hand, the pylorus is dragged from its moorings the lesser curvature dips obliquely from the left hypochondrium, passing even through the line of the navel; so that the stomach occupies the hypogastrium. The pylorus is thus bent upon itself, and the obstruction is indefinitely increased. The state of the coats of the stomach depends upon the character of the causes: if the dilatation be due rather to arrest than to failure, the reluctant muscular coat will tend to thicken, and with its increase there is usually an increase of the mucous coat also. If the hindrance be of slow growth, and the muscular coat undergo a parallel reinforcement, the cavity, as I have said, may not be enlarged, but may even be diminished; and the mucous coat, perhaps also thickened, may be thrown into exaggerated folds—the *état mamelonné*. Prof. Hamilton says: "The mucous membrane is thrown into innumerable pyramidal elevations with obtuse apices, each about the size of a lentil seed—probably caused by spasmodic contraction of the muscularis mucosæ, and of course it is often catarrhal." But in the vast majority of cases the cavity is greatly enlarged, and the hypertrophied muscular bundles lying between the connective-tissue septa—themselves also much increased—are very prominent. Unfortunately, as in compensatory hypertrophy elsewhere, the thickening tends to sclerosis. This process, probably identical with the fibrous condensation which we occasionally see in the pylorus itself, is irremediable. In chronic alcoholism this change is well seen, and in this case at any rate it begins in the submucous tissue, wherein, as well as in the proper muscular coat, the fibrous elements slowly advance at the expense of the functional elements. For obvious reasons the hypertrophy is first and chiefly seen about the pylorus, the main motor of the stomach, and there likewise the fibrous degeneration is conspicuous; we may still find muscular hypertrophy about the pylorus, when the muscularis of the fundus is atrophied by strain and pressure. There seems no doubt that in some cases the chronic and diffused thickening is of a carcinomatous nature, some leather-bottle stomachs being due to such an insidious infiltration; but as such changes do not fall within our present point of view we do but note them and pass on. That the leather-bottle stomach like a dense pylorus is by no means

always malignant my own experience can testify, and I am supported in this opinion by the records of Dr. Leith (p. 441), and of Drs. Perry and Shaw.

When we turn from these chronic and mechanical cases of gastrectasis to the acuter forms, or to forms which, if not very acute, are rather degenerative than mechanical in origin, we may fail to find compensatory hypertrophy. All the coats are thin, even wasted, as the partial obliteration of the secretory glands will show; and as the mucosa is almost surely the seat of a catarrhal process the muscularis is the less able to retain its natural elasticity. Mucus is found more or less abundantly in the cavity, and serves but too efficiently as the culture medium of yeast, sarcina, and other microbes, and as a source of some of the products of decomposition.

The contents of the stomach are excessively acid, and the reaction is usually due to the organic acids—lactic and butyric. It is alleged that hydrochloric acid is often present, even to excess; certain authors say, indeed, that in some cases hyperchlorhydria may be the efficient cause of dilatation; but the grounds for this belief seem insecure.

Diagnosis.—From what has gone before it is clear that the diagnosis of dilatation of the stomach must be made by looking at the body, by handling it, by puffing out the organ with an inflator, and, as subordinate means, by percussion with or without simultaneous auscultation. The physician will also have the help of the symptoms and history of the case. To these aids is to be added an investigation of the contents of the organ by syphonage—perhaps the most valuable means of all in cases which do not speak for themselves. Of this method I shall say more under the head of treatment (p. 510). The only final diagnosis of the malady is time after time to find considerable remnants of food in the viscus seven hours after a meal. A moderate meal is dispersed by that time, even by an atonic stomach; a light dinner is disposed of long before the lapse, say, of twelve hours. A progressive diminution in the average secretion of urine and desiccation of the tissues would corroborate the diagnosis, and suggest a tight pylorus. If bile be found in the stomach the pylorus is probably open. Dilatation due to mechanical obstruction is usually a persistent and inexorable process; that of atony remittent and mitigable. In obstructive cases peristalsis is more likely to become visible. In gastroptosis the lower border of the viscus is or may be manifest; the upper border is not to be found: I may remind the reader that a fallen stomach is not necessarily a dilated one, though it is in great danger of becoming so by kinking in the pyloric region. In ptosis other viscera, such as the liver and kidney, may also be displaced. Illumination of the stomach from within, as practised by Einhorn and by Reichmann and Herying (gastrodiaphany), does not seem to promise much so far. I admit that I have no personal knowledge of the method, still I am indisposed to believe, as Martens and Meltzing assert, that the full stomach under normal circumstances usually descends to the navel; it may do so, but it seems much more likely that the rays undergo so much diffusion that the method is fallacious. If substantial tumours occupy the stomach their

opacity to the rays may lead to their definition, scarcely to their discovery.

The absorption methods are of rather more practical value. In dilated stomach the rate of absorption is almost always slackened, usually very much so. If to such a patient potassium iodide be administered, the drug, instead of appearing in the urine in some ten minutes, will take an hour and more to present itself. Chlorides, again, abundant enough in the stomach, are scanty in the urine. In using such tests as these it must be remembered, first, that some of the solution may have passed through the pylorus; and, secondly, that absorption is retarded in states other than dilatation of the organ. Sidney Martin says, indeed, that the salol test is deceitful.

If the stomach be very capacious its distension is probably due to mechanical causes; if œdema of the feet be present, and free hydrochloric acid be absent or very scanty, the mechanical cause is probably cancerous. On the other hand, an atonic stomach, virtually dilated, may recover something like its normal limits when containing dregs only. Syphonage will then settle the question. The diagnosis of malignant destruction, if no tumour be palpable, must depend on the age of the patient, on his clinical history, and in some degree on the estimation of free hydrochloric acid. Of the lactic acid test of Boas I can say nothing; but it does not seem to gain general acceptance.

For some remarks on the diagnosis of dilatation due to the scar of ulcer the reader is referred to Dr. Dreschfeld's article on "Ulcer of the Stomach."

The following two cases are good examples of the occasional difficulty of diagnosis:—Hayem reports a case of a man, aged forty-two, who was admitted suffering from dyspnœa, cyanosis, cold extremities, and œdema of the legs. A rasping murmur was heard in the 4th intercostal space; and at the apex was heard a humming bruit like a mill-wheel, and not synchronous with the heart sounds. The urine was very scanty, but contained no albumin. The abdomen was distended, very painful, and tight, as if peritonitis were present. The stomach was now found dilated to a line some inches below the navel. Lavage was used daily with great relief; the urine was trebled in amount, and the cardiac murmurs ceased. After death stenosis of a non-malignant kind was discovered at the pylorus; in this case it would seem that the stomach arrested the diaphragmatic movement, pushed the heart upwards, and even deformed the chambers.

The second case I saw with Dr. Laurence Humphry. A middle-aged lady fainted, fell into collapse, and died within twenty-four hours. A thorough examination of the patient was impossible, but we detected, as we thought, considerable dilatation of the stomach. A good tossing splash was readily obtained, there was a tympanitic area and a definite cushion-like tumidity. On post-mortem examination this bag proved to be an enormously dilated transverse colon; the stomach was small and retracted, and lay high up under the ribs.

Prognosis.—This must depend greatly upon the causes and the degree of the dilatation, and no less upon the date of the diagnosis. In my experience, even if we exclude the cases secondary to stenosis, dilatation of the stomach is a very obstinate and generally an incurable malady. If there be stenosis of malignant origin the prognosis will fall under that of the primary disease. If the stenosis be not malignant, yet irremovable, relief of the symptoms is all we can hope for. In atonic dilatation without stenosis the prognosis, as a rule, ought to be hopeful; but, unless his diagnosis be made in very good time, the physician has no easy task before him. The treatment is very troublesome and in some respects very unpleasant; not only so, but with every advantage, as in cases of atonic dilatation after exhausting disease, if this state of the stomach become a substantial complication, the cure of it will be a long and difficult business.

Treatment.—We have to deal with an overworked and irritated organ becoming insufficient; its secretions and its motor activity fail; food is delayed in the cavity and the viscus dilates. How is this chain of events to be prevented? In dilatation consequent on stenosis there may be no loss of gastric juice, and the motor effect may be a relative one only. In these cases Kussmaul's lavage has enabled us to be of much service to a class of great sufferers. After a few test meals of cold meat and bread the physician will be able to ascertain the quantity and value of the gastric juice, and to estimate the digestive capacity. The necessity of syphonage will also be determined by the amount of residue found in the cavity at definite intervals. If bacterial fermentation be active, carbohydrates, such as starch, dextrin and sugars, must be withheld, or given sparingly. The syphon will also reveal the amount of catarrh, if catarrh be present. The conditions of the case being thus ascertained, the dietary can be arranged. The first rule, and one good for all cases, is that liquids by the mouth shall be restricted, if not forbidden; at any rate during digestion. Von Mering asserts that much of the fluid swallowed with a meal flows away in gushes through the pylorus, to be absorbed elsewhere; and Edkins (32) is quoted by Martin to the same effect. If, therefore, the pylorus be narrow or impassable, the ingestion of liquids adds a useless burden to the stomach already oppressed; and, by diluting the gastric juice, its digestive powers are still further enfeebled. The thirst itself, indeed, is sooner relieved by injecting liquids into the rectum.

Not only then must the diet be dry, but it must be administered finely divided in small quantities, and as economically as possible; that is, it must be concentrated, digestible, and nutritious. The use of milk in these cases is therefore not without great disadvantages; if, however, auto-intoxication be suspected, and milk in this respect desirable, one of the best brands of condensed milk without sugar may be prescribed. Otherwise tender meat, lightly cooked and teased from the fibre with a fork, is lighter, more digestible, and more sustaining. Meat essences, the dry meat powders, jellies, minces, and purées of chicken, fish and other tender meats may be allowed. Fat is so important an element

of diet, especially in emaciated patients, that unless the butyric fermentation be very active, some fats must be included in the diet. Eggs—raw, boiled, or in custards—are nutritious and of small bulk. Cream, again, or a little fresh butter, may be both pleasant and useful. In the cases of stenosis in which the gastric juices are not much in defect, carbohydrates may be taken in small quantity without creating the flatulence which is so mischievous in increasing distension at the very time when the stomach should be contracting. Vegetables are better avoided; if fresh meat be taken they are not necessary, or a little lemon juice may be taken occasionally. Untoasted bread should be forbidden, and likewise such starchy foods as arrowroot, sago, rice, and the like. In the earlier stages it is better to limit the starchy food to rusks and plain biscuits; but as the patient's state improves, malt extract may be added to the diet with advantage, as it is readily soluble, nutritious, and laxative. Liquids cannot be forbidden altogether. Small quantities of good old spirit, such as whisky or cognac, may be taken with a little water. Tea and coffee must be laid aside, but a little cocoa made from the nibs may be grudgingly permitted.

The quantity of food to be taken at each meal must depend upon the weight of the patient and upon his state of health; it should be divided into six small meals a day taken at nearly equal intervals. The fluid withheld from the stomach must be injected into the rectum. About half a pint of water, warmed to blood-heat, may be thus used three or four times a day; a little table salt, and, if needful, a little brandy, may be added to the injection. After a few days the rectum will accustom itself to receive the charge; but at first the fluid must be injected very gradually, a napkin must be pressed to the seat for a few minutes, and the patient should lie down on his left side until the sense of bearing down has ceased. In a day or two the urinary secretion will be augmented, it will contain chlorides more abundantly, and the tissues will become softer and plumper.

In atonic dilatation even more care in diet will be needed. In first undertaking an acute case of this kind, it is well to feed entirely by the rectum for ten days or a fortnight, with the usual precautions. A successful case was lately reported in which the patient lived for some long time and gained ground on chewing meat which was spat out when fully masticated. The patient was occupied all day long in chewing his cud. In the atonic cases, in which the gastric juice is more or less suppressed, malt extracts and peptonised foods are very useful.

It is better not to give very tasty things even if they seem digestible; the abnormal delay of food in the stomach makes flavours, not unpleasant in themselves, distasteful by their persistence. For this reason I do not order cod-liver oil, although it is a valuable remedy, and administered by many physicians in these cases. The rectum has some considerable power of taking up fat, and fresh suet smoothly mixed with milk may be given as an enema. The patient must be weighed regularly at the same hour of the day so that the fitness of the diet may be controlled.

Syphonage.—Both from the point of view of treatment and of diagnosis

lavage of the stomach is a most valuable agent. The information which it gives to the physician is often no less than the relief it gives to the patient. Not only so, but familiarity with the practice has led us to extend its use to the investigation of dyspepsias other than gastrectasis. Disagreeable and even revolting as it appeared at first, the remedy is now quietly submitted to; physicians and patients become less shy of strange methods, as horses get used to locomotives and bicycles: yet when, on the first appearance of Kussmaul's paper, I begged a lady of atonic fibre, afflicted with gastrectasis due largely to an abuse of aerated waters, to allow me to wash the stomach out, I begged in vain. Even hospital patients resented it at first; but at the present day men and women of refinement take to lavage as naturally as after a like period of shyness they did to morphia injections. As in the latter case, the danger may now be lest the relief be sought indiscriminately. In 1869 the stomach-pump was the means of lavage; soon afterwards a syphon, such as is now used, was made for me by Messrs Harvey and Reynolds of Leeds, and the same improvement soon suggested itself to other physicians. The stomach-syphon is too familiar now to need minute description: it consists of a tube of rubber, or still better of a thinner woven material like that of which flexible catheters are made, with a terminal or lateral opening at the distal end, large enough not to get blocked by solid matters; to the other end a large glass funnel is affixed.[1] It is well to introduce a short piece of glass tubing in the upper part of the length of the tube wherein the movements of the contents can be readily observed. These syphons are now sold by all surgical instrument makers. My first syphoned patient, or one of my first, was admitted into the Leeds Infirmary with benignant pyloric stenosis and large dilatation. Him I relieved so greatly by lavage that, being a sharp and inquisitive person, he set up as a quack on the strength of his experience, and washed out all the queasy stomachs of the country side. I believe he made a very good thing of it; so much so that, being a grateful as well as an ingenious person, he offered to give me a share of the booty. His irregular practice had the advantage of proving to the public that the process is less repulsive than at first sight it appears.

The syphon is a much safer and more convenient weapon than the pump. Twice, at least, I confess to having sucked off a portion of the gastric mucosa, and I have seen it done yet more frequently; indeed the accident has occurred often enough. No ill consequences seem to follow; still it is a piece of clumsiness which is rather discreditable. With the syphon this accident is by no means impossible, but it is less likely to happen.

I have almost given up the use of disinfectants in lavage. If the cavity be washed until the water returns clear, our purpose is sufficiently attained. The stomach never was and never will be aseptic; probably it is not desirable that it should be. On the other hand, the use

[1] Some physicians still recommend a rose. I used a rose in my first cases, but finding it very readily blocked up I did away with it. The tube should be lubricated with glycerine. [*Vide* "Diseases of the Œsophagus," p. 361 of this volume.]

of antiseptics may have its own dangers. If thick mucus be present the water should be made alkaline.

The purpose of syphonage is not only to clear the stomach of decomposed food, but also to cleanse it from the slime of catarrh, and from other fluids which seem to accumulate in larger quantity than food and drink can account for. For this reason, if the syphonage be practised, as is customary, once only in twenty-four hours, I always recommend it to be done in the morning before the digestive labours of the day are begun. It is a common practice to wash out at night, but a morning wash out will demonstrate that a great deal of tainted stuff finds its way into the sac during the night. Moreover, the results of the morning syphonage are a useful measure of the results of treatment. Sometimes there is great difficulty in emptying the stomach on account of the presence of solid or semi-solid matters which block the eye of the instrument. This trouble is generally worst at first; it tends to disappear after the diet has been carefully regulated, and the state of the cavity ameliorated. If the difficulty be considerable, it is well to insert an elastic ball in the tube in the place of the piece of glass tube; thus carefully a pumping action may be added to the syphonage, and plugs may be gently forced one way or the other.

It is scarcely necessary to say that if cancerous or simple ulcer exist, the tube should be used with the greatest care, or not at all.

Unpleasant as the process must be, such is the relief of the pain and other symptoms, both local and general, that patients often carry out the method with unfailing regularity, even after the vigilance of the physician is relaxed or his attendance has ceased. Generally speaking, one syphonage in twenty-four hours is sufficient; sometimes twice or thrice a week may suffice; on the other hand, in some bad cases a second cleansing in the later day may be desirable: the frequency of the operation will depend on the amount of fermentation. It is needless to say that, as a rule, four hours at least must elapse between a meal and the operation. In many cases of phthisis pulmonalis, probably cases in which the stomach is dilated, syphonage once or twice a day is of signal service.

As a rule the water used in syphonage must be lukewarm; but at the discretion of the physician a final dash of cold water may be given as a tonic to stomachs which retain some power of reaction. I may repeat that if there be much catarrh, a little bicarbonate of soda should be added to the water to reduce the tenacity of the mucus. The irrigation must be continued until the water comes away clear. Osler says that it is not necessary to remove all the water; it has always been my practice to remove as much as possible. Sidney Martin advises that after syphonage the funnel be removed and pressure made upon the epigastrium to expel the residual contents: while this is done the patient should lie on his left side. I dare say that this is good advice, at any rate in cases of pyloric stenosis when even clean water may not run readily away into the duodenum. An ingenious American physician showed to us one day, at

the Cambridge Museums, a hollow gastric sound armed with a revolving brush which by means of a handle could be thrust from the tube after its introduction, and whirled rapidly as the instrument travelled over the walls of the stomach. He assured us that this ramonage not only cleansed the walls but also invigorated them.

Wonderful as are the immediate effects of systematic lavage, yet as a cure it is often very disappointing. If, indeed, the ectasis depend upon permanent conditions, we cannot expect to bring about a cure unless the cause of distension be removed, and this is not often possible. On the other hand, when the ectasis is due to atony and catarrh, some considerable success ought to be attained. Now the difficulty in attaining anything like a reformation of the stomach seems to lie in the vicious circle between digestion and nutrition. The stomach suffers directly from its own insufficiency; and by failing to elaborate the means of repair it closes the door to its own restoration. To promote some advance in nutrition, I have used massage in a more or less irregular way.

In cases of extreme peril when collapse or exhaustion are present or imminent, or again in the last stages of organic disease, the introduction of the tube is to be deprecated. The operation itself increases the distress, and the results of syphonage are altogether disappointing; the stomach does not respond.

Massage is easier to obtain than it was, but it remains as yet the advantage of a few. Still I have seen enough of it in gastrectasis to expect much from the method; and in two cases I witnessed something like a cure by its means. In other cases improvement appeared; but, some months after leaving off the remedy, deterioration set in, though not to the former degree. From time to time, also, I read of the successful treatment of gastrectasis by massage in the practice of other physicians. An incidental advantage of the massage treatment is the efficient control over the patient and all his ways. In the treatise on massage by Dr. Eccles, I find that this author testifies to the value of massage in atonic dilatation. Dr. Eccles has discovered that many cases of alleged neurasthenia "have their foundation" in dyspepsia, using this word in a general sense—a truth which I hope to prove in the chapter on that disease. Massage is said to be potent to restore the qualities of the gastric juice; and cases of ectasis are said thus, and by improvement of general nutrition, to gain much by massage, which is calculated not only to promote nutrition, but also to dispel any toxic matters which would otherwise lie in the body. I have no doubt that massage is of service in cases of dilatation of the stomach of atonic kind; though I have yet to be assured that it can have any direct or considerable influence in pulling the organ together, or in evacuating its contents as is alleged by Zabludowski. Still its indirect advantages are no doubt substantial, and its influence upon the constipation is favourable. I have seen "rectal feeding and massage" proclaimed as a cure for simple dilatation. I must warn the reader that a patient on rectal feeding only will bear little massage or none.

Electricity may be found useful some day in atonic cases, but as yet I have seen no definite good from its use, whether internal or external; other physicians seem to have been more fortunate. The same is true, so far as my experience goes, concerning hydropathic means; so far, indeed, as regards those means which form part of the routine of water cures I am disposed to say that they are injurious.

Drugs.—The use of drugs to stop vomiting, in so far as they are successful, is probably mischievous. Happily it is not easy to produce the effect so unwisely desired. Vomiting, nature's means of relief, is rendered unnecessary by lavage. That gastrectasis is often or even occasionally due to hyperchlorhydria I must venture to doubt; at any rate I have not met with any clear instances of this sequence. I am strongly opposed, therefore, to the administration of large doses of alkalies and alkaline earths to neutralise the supposed hyperacidity of mineral origin, and at the same time to reduce the value of such mineral acid as may be needed. If the stenosis or the atony be not invincible, saline laxatives, such as Carlsbad salts or plain sulphate of soda, are of great service; but the remedy is to be used discreetly. A draught of ten minims of hydrochloric acid (B.P.), with a teaspoonful of the glycerine of pepsine and five minims of the liquor strychninæ, is to be given after each meal to a male adult. Takadiastase (gr. 3–5) is said to be potent in the digestion of starch, and in this sense may be an antacid. Some physicians speak well of papain in cases of defective secretion. In severe cases, at any rate for a time, some of the food should be predigested; I think, however, that this aid should be withdrawn as the stomach is enabled to do a little of its own work. Of other drugs nitrate of silver in pills is the most useful. Broadbent recommends creasote. None of these drugs is of any use unless the stomach be regularly washed out, and in any case absorption of the remedy, if this be desired, is slow and uncertain.

During the first institution of the treatment the patient must be kept in bed altogether, and bed must be continued if massage be systematically used; otherwise a little gentle exercise and fresh air within the patient's power are to be recommended. The warmth of the body is to be economised; and these very chilly patients had better keep their beds until some part of their heat-generating power is recovered, and the action of the heart invigorated.

In some cases of pyloric stenosis gastro-enterostomy has been performed with remarkable success. Operative interference, probably by gastro-enterostomy, would therefore be indicated in cases of persistent dilatation which does not yield to diet, medicine, massage and electricity. Gastro-enterostomy is said to be successful in simple dilatation, and to prolong life even in cases of slow malignant disease. Dr. Lauder Brunton tells me that he has had seven cases of gastro-enterostomy, three of which were successful, and one partially so. Dr. Walker of Peterborough reported such a success to the Cambridge Medical Society in 1896. On this subject Dreydorff's *Essay on the Surgery of the Stomach* may be

consulted with advantage. Almost as I write Dr. Ewart and Mr. Bennett publish in the *British Medical Journal* a case in which a reef was taken in on a dilated stomach; at least with temporary success. So long, however, as the stomach is in a state of catarrh and debased function, it is difficult to see that a severe operation can well be undertaken. If, however, the physician can restore some tone to the stomach and the heart then the surgeon may be in a position to tackle the obstruction with more confidence.

T. CLIFFORD ALLBUTT.

REFERENCES

1. ALLBUTT, T. C. *Lancet*, 1885, vol. ii.—2. ANDRAL. *Clin. Med.* vol. ii. p. 122.—3. BAMBERGER. "Tetanie bei Magendilatation," *Bericht. der Krankenanstalt Rudolfstiftung.* Wien, 1891.—4. BOAS. "Ueber Duodenalstenosen," *Berl. klin. Woch.* 1891, S. 949.—5. *Idem. Diagnostic u. Therapie der Magenkrankheiten.* Leipzig, 1893.—6. *Idem. Deutsch. mediz. Wochenschr.* 1894, No. 8.—6*a*. *Centralb. f. inn. Med.* Jan. 19, 1895.—7. BOUCHARD. *Leçons sur les intoxications*, 1887.—8. *Idem. Thérap. des maladies infectieuses*, 1889, p. 282.—9. BOUVERET and DÉVIC. *Rev. de méd.* Jan. and Feb. 1892.—10. BROADBENT, Sir W. H. On "Dilatation of the Stomach," *Brit. Med. Journ.* Dec. 2, 1893.—11. BUNGE. *Lehrbuch d. phys. u. path. Chemie*, Leipzig, S. 140.—12. COATS, J. *Manual of Pathol.* 1889.—13. COMBY. *Arch. gén. de méd.* 1884.—13*a*. DREYDORFF. "Kasuistischer Beitrag z. Magen-Chirurgie," *Beiträge z. klin. Chirurgie*, Bd. xi. Heft. 2.—14. DUPLAY. *Archives générales de méd.* 1833.—15. ECCLES, A. S. *Practice of Massage*, 1895.—16. EDKINS. *Journ. Physiol. Cambridge*, vol. xiii. p. 445.—17. EINHORN. "Ueber elekt. Magen und Darmdurchleuchtung," *Therap. Monatsheft*, 1892, S. 128.—18. ENDMANN. *Virch. Arch.* Bd. xlviii.—19. EWALD. *Congress für innere Medicin*, 1893.—20. FAGGE, HILTON. *Guy's Hospital Reports*, 1872-3.—21. FENWICK, SOLTAU. "Dangers of washing out the Stomach," *The Practitioner*, vol. xlviii. p. 241.—22. *Idem. The Dyspepsia of Phthisis.* London, 1894.—23. FOX, WILSON. Reynolds' *System of Medicine*, 1878.—24. FRANK, J. P. *De cur. hom. morb. epit.* lib. v. par. vi. s. 666, quoted by Penzoldt.—25. HAMILTON, D. J. *Text-Book of Pathology*, vol. ii. p. 504.—26. HAYEM. *Med. Mod.* No. 56; 1895.—27. HOPPE-SEYLER. "Untersuch. über Magengahrung," *Prager med. Wochenschr.* No. 19; 1892.—28. HUMBY and MILLER. *Path. Society Trans.* vol. iv. p. 137.—29. KUNDRAT. *Handbuch d. Kinderkrank.* Bd. iv.—30. KUSSMAUL. *Arch. f. klin. Med.* Bd. vi. p. 455.—31. LOEB. "Tetanie bei Magenerweiterung," *Arch. f. klin. Med.* Bd. xlvi. p. 95, 1889.—32. MARTIN, SIDNEY. *Diseases of the Stomach*, 1895.—33. MERING, VON. "Ueber die Functionen des Magens," *Verhandl. des XII. Congress f. innere Medicin zu Wiesbaden*, 1893.—34. MORITZ. *Münch. med. Wochens.* Dec. 3, 1895.—35. MÜLLER. "Tetanie bei Dilat. Vent.," *Charité-Annalen*, Bd. xiii. S. 273; 1888.—36. NOORDEN, VON. *Lehrbuch d. Pathologie d. Stoffwechsels.* Berlin, 1893.—37. OSER. Art. "Magenerweiterung," Eulenburg's *Realencyclopädie.*—38. PACANOWSKI. "Zur physical. Diagnostic d. mechan. Insuff. d. Magens," quoted by Osler, *Princ. and Pract. of Med.*—39. PEEBLES. *Edin. Monthly Jour.* 1840, vol. liv.—40. PENZOLDT. *Die Magenerweiterung.* Erlangen, 1875.—41. PERRY and SHAW. *Guy's Hosp. Rep.* vol. xlviii. 1891.—42. PEPPER and STENGEL. "Diagnosis of Dilatation of Stomach," *Amer. Jour. of Med. Sci.* Jan. 1897.—43. RIEGEL. *Deutsch. med. Wochen.* April 12, 1894.—44. REICHMANN and HERYING. "Ueber Gastrodiaphanie," *Berl. klin. Wochen.* 1892, Nos. 39, 40.—45. ROSENBACH. Volkmann's *Sammlung*, No. 153.—46. SENATOR. *Berlin. klin. Woch.* 1868, No. 24.—47. STRAUSS. *Berl. klin. Woch.* May 4, 1896.—48. SUTTON, H. G. *Lectures on Pathology*, 1891.—49. TAPPEINER. "Ueber Resorption im Magen," *Zeitsch. f. Biologie*, Bd. xvi. S. 497.—50. TROUSSEAU. *Clinique médicale*, t. ii. p. 347 (ed. 1862.)—51. WEGELE. *Die atonische Magenerweiterung u. ihre Behandlung.* München, 1894.

T. C. A.

ULCER OF THE STOMACH

SYNONYMS.—*Perforating ulcer, Simple ulcer, Round ulcer, Peptic ulcer.*

Definition.—An affection of the stomach clinically characterised by localised pain, vomiting, and hæmatemesis; and pathologically by a circumscribed loss of substance of one or more coats of the stomach by a process which appears to be a necrobiosis.

History.—Gastric ulcer seems to have been known to the ancients. It was known to Celsus, who describes ulceration which occasioned death either by perforation or by profuse hæmorrhage, and who even prescribes a treatment for gastric ulcer. Grassius records a case of perforation from gastric ulcer with discharge of the contents of the stomach into the abdominal cavity. Matthew Baillie, who devotes a chapter to the description of the appearance of gastric ulcer, speaks of its peculiar anatomical character as if "cut out with a knife." Abercrombie not only describes the anatomical character, but gives also the clinical features of the disease. An accurate description of the ulcer and its distinction from cancerous ulcers was first definitely given by Cruveilhier; he was soon followed, however, by Rokitansky, who looked upon the ulcer as a form of necrosis. The pathology of the ulcer was further studied by Virchow, and a few years later by Dr. Pavy; whilst the clinical features were delineated, amongst others, by Bamberger, Budd, and Brinton; Brinton's treatise, excellent both as regards statistics and exact clinical observation, is still quoted in most modern work on the subject. The application of the stomach-tube in the study of diseases of the stomach by Leube and Kussmaul has also furthered the diagnosis of ulcer of the stomach, which was advanced yet again by the more recent observations on the composition of the gastric juice and the discovery of clinical tests for the easy detection of hydrochloric and lactic acids in the gastric juice. Amongst the many observers who have advanced our knowledge on this subject I may especially mention Van der Velden, Riegel, Ewald, Galliard, Boas, Jaworski, Letulle, Debove, and Hayem.

A short summary of the history of gastric ulcer would not be complete without saying that the treatment of this disease has benefited by the advance of abdominal surgery, both in the relief of contraction of the pylorus and in the treatment of perforation of the ulcer.

Etiology.—Gastric ulcer is a common disease, but in respect of its frequency statistical records differ somewhat. Dietrich examined a total of 10,103 autopsies, and found open ulcers in 126, and cicatrised ulcers in 224—a percentage of about 3·4 of all autopsies; Brinton gives the percentage as 5; and this agrees with the figures given by Welch

from very extensive records: others give percentages as large as from 10 to 13.

Sex.—Gastric ulcer occurs more frequently in woman than in man. The proportion generally given is 2 to 1; some give it as 3 to 1. Welch, who collected 1699 cases of gastric ulcer from hospital post-mortem records, found that 40 per cent occurred in men and 60 in women. The post-mortem statistics of the Manchester Royal Infirmary give a large excess (2·5) to women.

Age.—Generally speaking, gastric ulcer occurs most frequently between the ages of 20 and 40. In women it occurs more frequently between 20 and 30; in men between 30 and 40. According to Brinton, who collected 226 cases of ulcers and scars, the frequency appears to increase, allowing for the number of persons living at the various ages, with advancing years; the numbers given by him are—

From the age of	0 to 10	10 to 20	20 to 30	30 to 40	40 to 50	50 to 60	60 to 70	70 to 80	80 to 90
Number of cases .	2	18	45	39	38	32	32	15	5

The conclusion of Brinton, however, does not accord with clinical observation; for he takes his numbers from post-mortem records, and probably in many cases the ulcer had existed for many years and had become cicatrised. If, instead of taking the post-mortem records, we select well-marked clinical cases, the larger proportion of cases in women will be found between the ages of 20 and 30; in men between 30 and 40.

Dr. Sidney Martin finds that out of 171 cases collected from the records of University College Hospital, where the age frequency was compared with the duration of the symptoms, 15 cases occurred between the ages of 0 and 10; 75 between the ages of 20 and 30; 38 between 30 and 40; 25 between 40 and 50; 14 between 50 and 60; and 4 in persons over 60.

Before the age of puberty gastric ulcer is of very rare occurrence; yet it has been observed in infants and occasionally soon after birth. The melæna of the newly-born, though often due to erosions and small multiple ulcerations, has in some cases been found to be due to simple ulcers of the stomach with regular, well-defined borders, varying in diameter from 1 to 3 centimetres, and situated in the stomach or duodenum (23). In other cases of melæna neonatorum no lesion has been found in either the stomach or intestines (26*a*).

Race and Climate.—From statistical records the disease appears to be equally common in the various countries of Europe, though in some parts it appears less frequent than in others; thus, according to Von Sohlern, it is very rare in some districts of Russia and in the Bavarian Alps. According to Da Costa (14*a*), it is less frequently found in America than in Europe.

Occupation.—According to Bamberger gastric ulcer occurs more frequently in cooks, and he attributes this to their habit of tasting food when it is very hot. From our own hospital statistics it appears to be more common in housemaids. Eichorst states that it occurs frequently in metal-turners; and he thinks it is produced in them by the irritation of particles of dust which are swallowed. It is said to affect those workmen who exert direct mechanical pressure on the epigastrium—such as tailors, shoemakers, weavers (35*a*).

Hygiene and Habits.—It is said to be more common amongst the poorer classes, who take insufficient or badly-prepared food, and live in badly-ventilated, small rooms. If this be true, it is probably only in so far as an anæmic state of the system is produced by these various factors. The opinion of Lancereaux that it is more common in drunkards, especially in those who take ardent spirits, is not confirmed by our own observations; the hæmatemesis in these cases is due to chronic venous congestion of the stomach; an ulcer has rarely been found in them.

Associated diseases.—Gastric ulcer is said to be more frequently observed with certain diseases; notably chlorosis, amenorrhœa, tuberculosis, syphilis, malaria, scurvy, chronic valvular affections of the heart, chronic Bright's disease, septicæmia and pyæmia.

Of these various affections the only one that presents indisputable relations to ulcer is chlorosis; probably the alteration of the blood in chlorosis, together with the alleged congenital smallness of some of the vessels, may help in the formation of the necrotic ulcer: in animals rendered anæmic an ulcer of the stomach has occasionally been produced. That in many chlorotic girls gastric ulcers occur after the chlorosis has lasted some time is a well-established fact.

In tuberculosis and in syphilis ulcers in the stomach specific of these affections may arise; these are, however, exceedingly rare; they differ anatomically from the simple ulcer and probably in their symptoms also. There is no evidence that tuberculosis disposes to the round ulcer of the stomach; and, according to Brinton's statistics, ulcer of the stomach occurs no more frequently in tuberculous subjects than in others; on the other hand, however, gastric ulcer may dispose to tuberculosis.

According to recent statistics of some observers (48) gastric ulcer is not rarely found in syphilitic subjects; endarteritis, a common occurrence in syphilis, affecting the vessels of the stomach, may probably account for this. Lang states that 20 per cent of all cases of gastric ulcer occur in syphilitic subjects, but this certainly is a much higher percentage than I could make out from my own cases. According to some observers, such as Wagner, the gastric ulcers found in syphilitic subjects are the result of ulceration of gummatous tumours; but this is certainly much too exclusive a view: gumma does occur in the stomach at a late period in syphilis, and has been found after death (49, 9, 12), but it does not give rise to any definite symptoms unless it ulcerate. The appearance of the ulceration of a gumma is different from that of a peptic ulcer: it has not the punched-out character; the defect in the submucous layer is greater than in the

mucous layer; the ulcer is irregular; its border is flattened, and in the neighbourhood of the ulcer several small gummas are usually found (57). In a few cases profuse hæmatemesis and symptoms of gastric ulcer which withstood the ordinary treatment have yielded to an antisyphilitic treatment (70).

For an account of the various affections of the stomach due to syphilis the reader is referred to Neumann's recent work on Syphilis (57).

In septicæmia and pyæmia ulcers of the stomach may probably be due to septic embolism; they are acute and multiple.

Traumatism.—How far simple injury, such as blows, kicks, or falls on the epigastrium and abdomen, may produce simple ulcers of the stomach is still disputed ground. It does occasionally happen that a blow on the stomach, or sudden severe pressure on the epigastrium, gives rise to profuse hæmatemesis; and this may be followed by epigastric pain and vomiting of food for some weeks; but it is doubtful whether these symptoms be due to an ulcer of the stomach. In several cases of this nature I have seen, the symptoms gradually disappeared, and none of the symptoms characteristic of a chronic ulcer of the stomach remained. That occasionally an ulcer may thus arise is probable from the observations of Potain and others (18, 22). The case is different when the traumatic cause is an extensive burn of the skin. Though when symptoms of ulcer follow a burn, the seat of it is almost always in the duodenum, yet in a few cases the ulcer has been found in the stomach. The relation of duodenal ulcer to burns, and the frequency of its occurrence, will be considered later. Swallowing corrosive poisons has in a few instances been followed by the formation of a gastric ulcer (29).

Heredity.—Most authors are silent on this subject; a few who mention hereditary influence in the causation of gastric ulcer look upon the evidence as unsatisfactory. I have taken some pains to inquire into this matter. In a few cases the account of gastric ulcer in various members of the same family rested on insufficient grounds, but in eight cases the evidence appeared conclusive; in two instances mother and daughter suffered from it; in one, father and daughter; in two, two sisters; and in one instance brother and sister suffered from unmistakable symptoms of the disease.

Pathological anatomy.—Ulcers having the characteristic appearance of gastric ulcer are only found in the stomach, in the duodenum, and in the lowest part of the œsophagus.

Number.—Usually one ulcer only is found; occasionally two or even more may be found (according to Brinton, multiple ulcers are found in about one-fifth of the cases); if two are found, they are sometimes situated opposite to each other; more often they are close together, one generally being chronic or even cicatrised, the other of recent date.

Situation.—Most commonly the ulcer is found at the pylorus, more frequently on the posterior wall on or near the lesser curvature; occasionally we find it on the anterior wall (more often in acute than

in chronic ulceration); in rare instances the ulcer is situated on the greater curvature, and more rarely still on the fundus of the stomach.

Size.—The size of the ulcer varies. Generally it is a little less than an inch in diameter; but the diameter may be smaller, less than half an inch, or larger, measuring two inches or more. Small ulcers are usually acute ulcers. In rare instances, as in the cases recorded by Cruveilhier and by Law, ulcers much larger still have been found. In the case of a woman who died at the Manchester Infirmary, in a small stomach measuring about five inches from the cardiac end to the pylorus and only two inches in circumference at the broadest part—a stomach less than the transverse colon—an extensive cicatrix was found extending from the cardiac end to the pylorus; there was a history of profuse hæmatemesis fifteen years before death, but during her stay in the hospital the patient had no dyspeptic symptoms and could take the ordinary amount of food.

Appearance.—This varies according to the duration of the disease, and we may distinguish between *acute* ulcers of recent origin and *chronic* ulcers. The acute ulcer is usually round, occasionally oval; its characteristic appearance is as if it were punched out of the wall of the stomach; the edges are clean cut, smooth, not thickened; the floor is smooth, occasionally irregular, firm, or somewhat pulpy; in rare cases it may contain a slough of necrotic tissue. Small hæmorrhages and congestion may occasionally be found on the edges and in the neighbourhood of the ulcer. The depth of the ulcer varies; it may extend to the submucous, muscular, or even serous coats, and as each successive layer of the wall of the stomach may be less destroyed than the preceding layer, the ulcer may present a funnel-shaped appearance, or be like a terrace with sloping edges.

In ulcers of long standing the appearance is different. The ulcer is generally larger in size, round, oval, irregular, or occasionally, if of very long standing, girdle-shaped with signs of scarring. The edges are thickened, the funnel-shaped appearance is often well marked, and the floor either smooth or irregular. The floor is formed by the serous surface, which is thickened by fibrous deposit, the result of chronic inflammation. This exudation may lead to the adhesion of the ulcer to the adjacent portion of the peritoneum, and to the contiguous organ; thus most commonly we find adhesion of the stomach to the pancreas, as the ulcer is most frequently situated on the posterior wall of the stomach near the pylorus: occasionally we find adhesions between the liver and the stomach; and in some cases, if the tumour be situated near the cardiac end of the large curvature, the stomach may become adherent to the spleen, to the great mesentery, to the diaphragm, and so on.

In the course of time the new tissue may become absorbed, and the floor of the ulcer may then be formed by the neighbouring organ itself; as, for example, by the pancreas denuded of its peritoneal covering, or the liver, or the spleen. I saw one case in which the floor

of the ulcer was of an irregular and convex outline, and consisted of necrotic liver tissue; and further examination showed that the ulcer had eaten away a considerable mass of the adjacent portion of the liver. If the ulcer be situated on the anterior wall of the stomach, we notice, owing to the greater mobility of the part of the stomach involved, and the absence of any fixed organ in the neighbourhood, either no adhesions or very slight ones; hence it is that ulcers situated in this locality so often perforate.

Perforation may occur wherever the ulcer lies, if the adhesions be not very firm. The results of the perforation vary according to the size of the perforating opening and its situation. If the ulcer perforate into the peritoneal cavity, and if the opening be large, some of the contents of the stomach may pass into the peritoneal cavity and give rise to perforative peritonitis; if the opening be very minute, there may be but a local peritonitis and a formation of fresh adhesions by which the small opening may be occluded; if perforation take place into an adjacent portion of the intestine, a bimucous fistula is established; such a fistulous communication is most commonly formed between stomach and transverse colon, occasionally between the stomach and duodenum, or between the stomach and another portion of the small intestine; much rarer are fistulous tracts between the stomach and the pericardium, the pleura, the gall-bladder, or a bronchus. The inflammation set up may not always result in the formation of fibrous tissue, but may result in the formation of an *abscess*. In most cases, no doubt, the abscess is the result of a small perforation, as, for instance, the subphrenic abscess, or abscess between stomach and anterior wall; whether such an abscess may form without previous perforation is difficult to prove [*vide* art. "Subphrenic Abscess," p. 570].

Gastric ulcer being a form of necrosis, we can readily understand how in the course of the destructive process blood-vessels become eroded, and hæmorrhage, more or less profuse, occurs. This is as frequent a feature of the acute as of the chronic ulcer. If the arteries are examined microscopically, periarteritis and endarteritis are often found. This change, when the artery is small, leads to more or less diminution of the lumen of the vessel, and may end in the formation of a thrombus by which hæmorrhage is prevented. In other cases the walls of the small arteries are softened and transformed into embryonic tissue which may cause or aid the hæmorrhage so often observed in ulcers (16).

If a hæmorrhage be fatal we may often detect the open mouth of a fairly large artery in the centre of the floor of the ulcer. The microscopic examination of the vessel in one case showed scarcely any pathological change in the walls of the artery itself, except at the ruptured end, where a mass of granular detritus was found.

The acute and the chronic ulcer alike may undergo complete *cicatrisation;* if the ulcer has been superficial, not penetrating to the peritoneal coat, we notice only a slight depression in the mucous surface and

slight thinning of the wall of the stomach; if, however, the ulcer has penetrated to some depth, the cicatrix is formed of firm, fibrous tissue which often causes a distinct puckering of the neighbouring parts. The formation of the scar, though it may lead to the healing of the ulcer, may give rise to further trouble by mechanical obstruction to the passage of the food, the most common occurrence being stenosis, or narrowing of the pylorus, and subsequent dilatation of the stomach; or if the ulcer be situated near the pylorus on the large curvature, the stomach may be converted to the shape of an hour-glass. Ulcers in other situations may also lead to various displacements of the stomach, and to alterations in its size, appearance, and connection with other parts.

Histologically examined we do not find in the acute ulcers, either at the borders or in the floor of them, any changes other than destructive; in the chronic ulcers, however, we find signs of inflammation. The glands at the border of the ulcer appear enlarged and tortuous, some are changed into small cysts, in others the epithelium has undergone a marked change; we can no longer distinguish between the "Hauptzellen" and "Belegzellen"; the whole lumen is distended with small cubical and cylindrical cells, which also line the walls of the glands; the interglandular tissue is thickened and contains newly-formed fibrous tissue; cell infiltration and new formation of fibrous tissue is also found in the submucous and muscular layers. The floor of the ulcer presents similar appearances; the muscular layers are more or less completely replaced by fibrous tissue which, in ulcers of long standing, form dense fibrous layers.

Apart from these changes it would appear from the observations of Galliard and the more recent observations of Jaworski and Korczinski that the mucous membrane of the stomach in all cases of gastric ulcers shows changes which become more evident on microscopic examination. These changes consist in a cell infiltration between the several layers of the coats of the stomach and marked inflammatory changes in the walls of the blood-vessels (both of veins and arteries), and in the neighbourhood of the nerves also. Jaworski and Korczinski examined portions of the walls of the stomach removed on operation in cases of gastric ulcer, and found very similar changes, together with the degeneration and disintegration of the "Hauptzellen." These changes were found in sections taken from various parts of the mucous membrane of the stomach, even when a good distance away from the seat of the disease. According to these authors the inflammatory changes are constantly found in cases of gastric ulcer, and are looked upon by them as primary; but many pathologists look upon them as secondary, and as not always present.

Pathogenesis.—Our knowledge of the exact causation of the gastric ulcer is as yet far from definite, and many are the views held on the pathogeny of it; this is not surprising, as the ulcer is not always due to one and the same cause; in many cases, indeed, several causes operate together, some more immediate, some more remote.

Two factors stand out clearly when we consider the causation of gastric ulcer: the one is that the ulcer is not the result of inflammatory but of simple necrosis; the other is that, as the typical round ulcer is confined to the stomach and the adjacent portion of the duodenum or œsophagus, the gastric juice plays some essential part in its production.

There are many causes of necrosis, and thus the opinions on the causation of ulcer are likewise various.

Many believe that the chief cause of ulcer is an interference with the blood-supply of the affected portion of the wall of the stomach; if the nutrition of the part were thus seriously interfered with, a necrotic ulcer would be the result, with or without the help of the gastric juice. Interference with the blood-supply may take place in various ways; as by embolism and thrombosis of an artery, diseases of the coats of the blood-vessels (atheroma, amyloid degeneration, fatty degeneration), spasm of an artery, or compression of the veins by spasmodic condition of the muscular coat of the stomach resulting in a hæmorrhagic infiltration (Rindfleisch). Each of these causes has found its advocates. Virchow, after he had discovered the embolic process, attributed ulcer to embolism of an artery; and this doctrine has had many advocates since: but against it we may urge that it is only in extremely rare cases that an embolus has been found; and that in experiments on animals the injection of solid particles into the circulation has very rarely produced gastric ulcer (11, 59).

That the necrosis should be caused by thrombosis in an artery is also improbable, for thrombosis occurs chiefly in arteries whose walls are diseased; gastric ulcer, on the other hand, occurs much more frequently in young persons in whom degenerative and other changes in the walls of the blood-vessels are very uncommon.

Temporary spasm of the artery was supposed by Klebs to be a cause of such a deficiency of blood-supply as would lead to necrosis; but this view is purely hypothetical, and finds, indeed, few supporters: nor can more be said for the view, first expressed by Rokitansky, that venous hyperæmia is the direct cause.

It will be seen, then, that the above-mentioned conditions in arteries or veins can be but very occasional causes of gastric ulcers.

Other causes which may explain necrosis are:—

(i.) *Chemical and thermal.*—We can readily understand how such ulcers might be due to poisoning with corrosive substances, whether acids or alkalies; but this, after all, will explain but a very few isolated cases. It has been thought that scalding food might produce a limited amount of necrosis, and in this way the frequent occurrence of gastric ulcers in cooks has been explained. The experimental investigations of Decker (17*a*) supported this view, for by introducing very hot food by means of a stomach-tube into the stomachs of two dogs, he produced in one case marked hæmorrhagic erosion, and in the other a typical acute ulcer of the stomach. In a few cases gastric ulcer in man

may be produced in this way, but certainly only in a few; for slight loss of substance of the stomach wall heals readily, and even if a hæmorrhagic erosion should follow, it by no means always gives rise to gastric ulcer (Langerhans).

(ii.) *Mechanical.*—Continued pressure, such as produced by tight lacing or tight belts, may be an occasional cause. Rasmussen pointed out a distinct sulcus in the anterior wall of the stomach which passes vertically from the small to the large curvature close to the pylorus, and is opposite the lower line of ribs; this line of depression, which shows thickening of the serous coat, atrophy of the mucous surface, and occasionally peritonitic adhesions, he looks upon as due to pressure; and in several cases along this line he found scars of gastric ulcer.

(iii.) *Traumatic.*—I have already said, when speaking of the etiology of this disease, that injury may produce gastric ulcer. In animals Ritter and Vanni have studied the effects of blows on the epigastrium, single and repeated. In this way distinct ulcers were produced in some cases; in others, hæmorrhagic infarcts, or interstitial hæmorrhages, which latter were looked upon as giving rise to ulcer. The ulcer, however, had not the characteristic appearance of the round ulcer met with in man. Direct injury of the mucous membrane, as shown by the experiments of Quincke, and of Griffini and Vassale, does not give rise to the formation of ulcers; this may often be verified when on washing out the stomach large pieces of the mucous membrane are accidentally detached, for the defects caused by such injuries heal very readily.

(iv.) *Neurotic causes.*—Secretion, vascularity, and other functions of the stomach being dependent on many nervous influences, the nervous system is held by some authors to play an important part in the causation of a gastric ulcer. It may do so in various ways. We know that after injuries of the nervous system (8, 74, 83, 24), and sometimes also in diseases of the central nervous system, punctiform hæmorrhages or small hæmorrhagic erosions are found in the stomach. But, even if vaso-motor disturbances, leading to the small hæmorrhages, were more common, it is very doubtful whether small hæmorrhages or hæmorrhagic erosions lead to gastric ulcers. Wilks and Moxon regard the gastric ulcers in the light of trophic ulcers, like simple ulcer of the cornea; whilst others, such as Liebert and Gunsberg, think that owing to nervous influences a hyperacid gastric juice is secreted which, as we shall see presently, is looked upon as instrumental in the causation of gastric ulcer.

(v.) *Gastritis as a cause.*—The early writers on gastric ulcer, such as Abercrombie and Cruveilhier, looked upon chronic catarrh of the walls of the stomach as the primary cause of gastric ulcer; this view was abandoned after the vascular infarct hypothesis was proposed by Virchow and Rokitansky, but it has been revived by Galliard and by Jaworski and Korczinski, who, as already mentioned, found signs of gastritis in different portions of the stomach in cases of chronic ulcer.

Dr. Soltau Fenwick looks upon a morbid condition of the solitary glands as a likely cause of acute gastric ulcers; he produced ulcers in guinea-pigs by inoculation with pure culture of typhoid bacilli, and he found that the ulcers had taken their origin in solitary glands; he cites also the result of an examination of a specimen of gastric ulcer in man in which such an origin was made out.

(vi.) *Alterations of the blood* may act either as a direct or as a more remote cause. The profound alteration of the blood in hæmoglobinuria, as in cases recorded by Silbermann, may cause gastric ulcer by producing either hæmorrhagic erosions or capillary emboli. The acute ulcers formed after burns are probably caused in this way, as will be explained under the head of duodenal ulcers; other alterations of the blood, as in malaria and chlorosis, have a more remote effect, and act by virtue of a local anæmia or diminished nutrition.

Quincke and Daettwyler (64) found that after applying various mechanical and chemical irritants to the gastric mucous membrane in dogs rendered anæmic by repeated depletions, a loss of substance of the mucous membrane led to ulcers which did not readily heal.

Bacterial necrosis.—Boetcher found micro-organisms in the borders and floors of gastric ulcers. Letulle more recently expressed the opinion that some of the gastric ulcers may owe their origin to microbes. Gastric ulcers have been noticed in pyæmia, puerperal septicæmia, and other infectious diseases; more often, however, instead of typical gastric ulcers, hæmorrhagic erosions have been found, and microbes have been detected in them. Again, injection of pure cultivation of microbes, such as the staphylococcus pyogenes, into the stomach or into the peritoneum gives rise to hæmorrhagic erosions, and even to ulcerations. The relation of ulcerations in the stomach and intestinal tract to micro-organisms has recently been studied more closely, and it has been clearly demonstrated that ulcerations of the alimentary tract in animals may be caused by the invasion of various micro-organisms into the deeper layers of the mucous membrane and the lymphatic glands connected with it, where they set up necrosis (66). This fact has been made use of by Sidney Martin (54) to explain the formation of chronic gastric ulcer by bacterial necrosis; and the more frequent occurrence of gastric ulcer at the pyloric end he explains by the absence here of the glands in this locality which secrete hydrochloric acid, which has a powerful antiseptic action, and prevents the invasion and growth of bacteria. But as yet this view, so far as the gastric ulcer of man is concerned, may be looked upon as purely hypothetical.

The Gastric juice.—It will be seen from the above that many causes may lead to hæmorrhagic infiltration and so be the starting-point of the ulcer, but another factor plays a significant part in its complete formation and extension. Dr. Pavy was the first to draw attention to the auto-digestion of the stomach by its gastric juice, if the superficial layers, which form a protecting film, have disappeared, or if the supply of blood to a certain portion of the stomach be interfered with;

for the blood rendering the part alkaline neutralises the acid. Considering how quickly defects in the walls of the stomach heal over in man, and that the alkalinity of the blood may be neutralised without leading to ulceration (63), this view can scarcely be accepted. Yet that the gastric juice has some effect in the production of the ulcer is evident from the occurrence of the ulcer in those parts of the alimentary tract only with which the gastric juice comes into contact. As in most cases of gastric ulcer the gastric juice is hyperacid, the excess of hydrochloric acid is now looked upon by many as a condition favourable to the production of a gastric ulcer.

Riegel looked upon the hyperacidity as a constant concurrence; but this is certainly not the case as shown by the observations of C. Gerhardt and Rosenheim, and confirmed by my own observations: yet we may say that in a very large majority of ulcers the gastric juice is hyperacid. Mathes (55) by daily administration of hydrochloric acid succeeded in producing a gastric ulcer in a dog in which an artificial defect in the mucous membrane had been previously made.

Symptomatology.—The symptoms of gastric ulcer vary considerably; in some cases the ulcer may be entirely latent, or give rise to no characteristic signs; in others, especially the acute ulcers, the first marked symptom may be profuse hæmatemesis or the symptoms of acute perforation. In most cases, however, besides dyspeptic disturbances, we have certain symptoms which may be looked upon as characteristic; namely, localised pain in the epigastrium coming on at a variable period after food, vomiting and hæmatemesis. In long-standing cases, apart from these stomach symptoms, we often see signs of anæmia, of malnutrition and of emaciation.

If we consider the symptoms separately, we have to note:

Pain.—This is perhaps the most frequent symptom. The patient complains of pain situated mostly in the epigastrium, just below the ensiform cartilage; it is distinctly local, and usually covers but a small area; occasionally it starts at the epigastrium, and extends thence to the left or to the right; in very chronic cases the pain is often situated some distance below the epigastrium, and is more diffuse; in most cases, besides the epigastric pain, there is also pain in the back, either over the region of the spine between the sixth and eighth dorsal vertebræ, or to the left of the spine and then situated somewhat lower, between the tenth dorsal and first lumbar vertebræ. The pain varies in character and intensity; usually it is of heavy boring character, at other times it is described as a burning pain, in some cases as a shooting pain, and at times it may become much more intense, more like a colicky pain (gastralgia), and may lead to actual syncope; in the early stage the pain is usually much less severe. The pain is not continuous, it comes on after meals, sometimes very shortly after food is taken, and in this case probably the irritation by the solid food is the cause of it; at other times it occurs some time

after food; in not a few cases the pain occurs several hours after food has been taken; the movements of the stomach, the increasing acidity of the gastric juice, and the passage of the stomach contents through the pylorus are probably the cause of it. In some cases the pain occurs at night or early in the morning when the stomach is empty; then the hyperacidity of the gastric juice directly acting on the ulcerated surface is most likely the cause of it, for to take a small quantity of food often relieves this nocturnal pain. Palpation and slight pressure of the epigastrium increase the pain, and elicit it at the time when it does not occur spontaneously.

The duration of the pain varies; often it lasts for several hours, in fact till the contents of the stomach have passed the pylorus, or till the food has been vomited, when it more or less quickly disappears. The pain is also influenced by a variety of circumstances, as for instance by the posture of the patient; it is less when the patient lies down, and it is aggravated by sudden movement or by undue muscular exercise or fatigue; some patients find relief when they lie on the abdomen (according to Brinton, in these cases the ulcer is situated on the posterior wall of the stomach); menstruation and mental emotions often increase the pain likewise.

The spinal pain has the same character as the epigastric, and the patient often states that it passes right through from the epigastrium to the spine. With the spinal pain, which, as first pointed out by Ross and more recently by Head, is an associated pain, there is often pain along the intercostal nerves, occasionally radiating to the shoulder, arm and forearm. The situation of the pain, being so often a referred or associated pain, does not indicate the exact site of the ulcer, as some authors maintain.

The pain may be absent for days and even weeks, and then recur; with the cicatrisation of the ulcer it gradually disappears, but, though in these cases there is no spontaneous pain, it can be elicited, as a rule, by pressure on the epigastrium.

As gastric ulcer is often accompanied by dyspeptic symptoms, the patient, besides the local pain, will sometimes complain of a dull pain, which is more diffuse and persists for days. In some few cases pain is absent.

Hyperæsthesia of the skin over the epigastrium and upper part of the abdomen, as pointed out by Head, I have repeatedly found.

As in some cases of gastric ulcer pain may be almost the only symptom, and may be associated with vomiting of food or hæmatemesis, a careful inquiry as to the exact situation, limitation, and the other characters described above, is of great importance in diagnosis. For illustrative cases I refer the reader to my paper on the Clinical Diagnosis of Gastric Ulcer (21). Two of these cases are very interesting—one was the case of a lady's maid, whom I saw one morning, when she simply complained of pain after food, so severe that she had to lie down; but it soon passed off, and troubled

her otherwise so little that she was able to do her work; physical examination of the chest and abdomen revealed nothing abnormal except tenderness over a limited area in the epigastrium. I looked upon the case as one of chlorosis. On the evening of the same day I was sent for hurriedly to consult with Mr. W. Davies in a case of acute peritonitis. To my great astonishment I found the patient to be the lady's maid whom I had seen that morning. It appears that she left the house to spend the day with some friends, and on the way she was taken again with intense abdominal pain, which became so severe that she was obliged to take a cab and to drive to a relation living near. Arrived at this house she was obliged to go to bed, and the pain became so violent that Mr. Davies was sent for. When I saw the patient she was collapsed; the body covered with cold clammy perspiration; the pulse small, threadlike, 140 per minute; the abdomen tympanitic and painful to the touch: the liver dulness could not be made out. In fact, the patient had all the symptoms of perforative peritonitis. She died the same evening, and, though no post-mortem examination was made, there can be but little doubt, I think, that we had here to do with a gastric ulcer which perforated into the abdominal cavity. Another very striking case was as follows: a girl, aged 14, was brought to me by her mother, with all the symptoms of chlorosis; she complained of slight pain in the epigastrium coming on after food, and had most severe pain in the dorsal region of the spine; there had been some loss of appetite, but no vomiting; physical examination revealed nothing beyond the signs of chlorosis; the dorsal region of the spine was very tender to the touch, and the pain very much increased on walking. In spite of tonics and careful dieting there was no improvement; the pain in the back became so severe that a surgeon was consulted, who suspected spinal caries and ordered the patient to keep the recumbent posture. The pain, however, did not subside, and after a few months the patient had severe hæmatemesis and melæna and died. Evidently this also was a case of gastric ulcer.

Vomiting.—Vomiting of food alone, without hæmatemesis, occurs frequently in gastric ulcer; it may occur at various stages of the ulcer and be caused in various ways. It may come on early in the course of the ulcer, and occur immediately after taking food, in which case it is due most likely to increased instability or hyperæsthesia of the stomach. In other cases it is due to the irritation produced by the contact of the food with the ulcer; here the vomiting occurs an hour or two after the ingestion of food, and is preceded by pain, which disappears after the vomiting; whilst in other cases the vomiting comes on late in the disease, and is due to dilatation of the stomach in consequence of the pyloric constriction. The vomit itself may present some features which assist in the diagnosis of the disease. The vomiting that occurs in consequence of dilatation of the stomach is, as a rule, characteristic enough: large quantities are voided pale in appearance, not unlike barm, frothy, and microscopically showing torulæ and sarcinæ; the filtrate almost

always shows the presence of free hydrochloric acid and albumoses. In those cases in which there is no dilatation of the stomach, the vomit, if it occur immediately after the ingestion of food, consists of unaltered food; if it occur some time after food has been taken, the vomit consists of partly digested food, and has a very acid taste and acid reaction. The chemical examination of the vomit or its filtrate almost always shows the presence of hydrochloric acid, very often in more than the normal quantity, and of lactic acid; peptones and albumins are present as traces; sugar not often. The microscopic examination of the vomit shows the muscular fibres of the flesh food partly digested. The vomit in gastric ulcer rarely contains bile; the so-called "bilious vomiting" is a rare phenomenon in gastric ulcer.

As the vomiting of very acid matter, containing more than a normal amount of hydrochloric acid, occurs in a good many other affections (as in one form of acid dyspepsia, in nervous dyspepsia, in the early stage of phthisis, in the gastric crisis of locomotor ataxy), the other symptoms accompanying it are important to help us to arrive at a correct diagnosis.

Hæmatemesis is one of the cardinal symptoms, and occurs in about one-third of the cases. It may occasionally be one of the first symptoms noticed, and occur in persons who were either in good health without any symptoms of indigestion, or who were troubled only with so-called acidity of the stomach; more often it occurs during the course of the disease, and is preceded by pain and vomiting.

The quantity of blood vomited varies considerably; slight hæmorrhages may often occur, and small quantities of blood may be vomited without the patient noticing it; in other cases the blood may not be vomited but is passed out per rectum as melæna; in most cases, however, where the hæmorrhage from the ulcer is profuse, both hæmatemesis and melæna appear, the latter following some hours after the hæmorrhage. If melæna should occur before hæmatemesis, most authors look upon the case as one of duodenal rather than of gastric ulcer; but in a case of acute gastric ulcer which I recently saw—one verified by post-mortem examination—melæna had preceded hæmatemesis by more than six hours. If the quantities vomited be small, the blood, intimately mixed with the food, may escape detection; if it be slowly poured out from the ulcer, the blood may remain for some time in the stomach, and, being acted upon by the gastric juice, may have the "coffee ground" appearance, the dark brown colour being due to the formation of hæmatin from hæmoglobin. If the hæmorrhage from the ulcer be profuse, as for instance if a small artery be eroded, then blood is vomited in large masses: it may be quite liquid and unaltered, but occasionally it contains large clots, and, though arterial, is dark in colour from the action of the gastric juice. In profuse hæmatemesis the patient usually has, as a premonitory symptom, a peculiar sensation of distension; he has a feeling of heaviness in the stomach, he is sickly and faint, and has a peculiar taste in the mouth; then without any violent effort of retching large quantities of blood are vomited: the first

hæmorrhage is often followed by a second and third at various intervals. The general condition of the patient is that common to all profuse arterial hæmorrhages: the skin is pallid; the patient feels cold, though often the temperature, soon after the hæmorrhage, may be a few degrees raised; the pulse becomes quick, small, and compressible; the patient feels faint; his voice becomes feeble; and syncope may follow an attempt to rise from the recumbent position. These symptoms continue for some days, and anæmic bruits are heard over the heart and the large vessels.

Hæmorrhage from the stomach, even if profuse, is not often fatal, unless a large vessel be eroded, when death may occur even before there is time for the poured-out blood to be vomited. In some cases of profuse hæmorrhage several attacks of hæmatemesis occur, and the patient dies from exhaustion; in most cases, however, the patient gradually recovers from the anæmia, and the symptoms of gastric ulcer, such as the epigastric pain which may disappear after the hæmatemesis, return. As a rule, several hæmorrhages follow the first at intervals of a few days, and then the hæmorrhage may cease altogether or reappear at a later period.

Melæna.—We have already spoken of this symptom following profuse hæmatemesis; if the ulcer be situated close to the pylorus it may occur alone without hæmatemesis. The blood in melæna is black in colour and of tarry consistency owing to the change of hæmoglobin into hæmatin, and the formation of sulphide of iron by the action of the sulphuretted hydrogen in the lower bowels. This symptom has often to be inquired for, or can only be inferred, as the patient is not always conscious of having discharged blood: he may complain, however, of tenesmus and of having had several liquid stools; or again, that during and after the evacuation of the stool he felt faint or actually fainted; and in his appearance there may be all the signs of a profuse internal hæmorrhage.

In three of my own cases general convulsions occurred in consequence of melæna from gastric ulcer. When hæmatemesis occurs along with the other symptoms it helps to complete the diagnosis; if it be one of the first symptoms, and not accompanied by the others, it has to be distinguished from the hæmatemesis due to chronic venous congestion of the stomach, as in cirrhosis of the liver and chronic affections of the heart. In these cases, apart from the other symptoms present, the blood, being venous, is generally fluid and dark, and the prostration of the patient is by no means so great as in the hæmatemesis from gastric ulcer; the anæmic symptoms quickly disappear, in fact, the hæmorrhage may be compared to a large venesection. In hysteria hæmatemesis may occur, and as hysteria often complicates gastric ulcer in young women it is well to distinguish between these two forms of hæmatemesis: in hysteria the quantity of blood vomited is usually small, the hæmorrhage occurs chiefly in the morning before food is taken, and may well be due to the artifices of the patient, though

there can be no doubt that in some cases the blood actually comes from the stomach. The presence of other hysterical symptoms, such as hemi-anæsthesia, globus hystericus, hysterogenic zones, contraction of the field of vision, and so on, are important aids in the differential diagnosis.

Such rare cases of hæmatemesis as those due to bursting of an aneurysm into the œsophagus, or into the stomach, are quickly fatal; hæmatemesis from an ulcer of the stomach is rarely followed by death.

In cancer of the stomach hæmatemesis is never, so far as my experience goes, the first symptom; the vomit has more of the coffee-ground character, and the other symptoms — emaciation, cachexia, tumour—are generally present. Hæmatemesis due to blows and other traumatic causes we have already considered.

Apart from these principal symptoms in gastric ulcer we have others less characteristic and less frequent. Considering these symptoms according to the various systems we have:—

Gastro-intestinal symptoms.—The tongue varies very much in appearance; often it is red, clean, and moist: when there is marked anæmia it is pale and flabby; in chronic cases, on the other hand, it is often found covered with white fur, which is due to the accompanying gastric catarrh. The appetite is generally good, but the patient is afraid to eat because of the pain after food; sometimes the patient suffers from an increased appetite; in very chronic cases the appetite is often diminished. Thirst is increased, and in some cases there is an increased flow of saliva. Dyspeptic symptoms, such as flatulence, sense of fulness and of discomfort after food, acidity and pyrosis, are common, especially in chronic ulcers; constipation is usual, but in a few cases the bowels are regular; diarrhœa is very rare.

Cardio-vascular symptoms.—In many cases nothing abnormal in the heart or blood-vessels is noticed; occasionally there is irregularity of the heart's action, and, as anæmia so often complicates gastric ulcer, we have all the vascular symptoms noticed in anæmia.

Genito-urinary symptoms.—The urine is in many cases normal, at other times it may contain albumoses; on the other hand, owing to the increased secretion of hydrochloric acid, it is often but slightly acid and may even become alkaline; this is especially the case when the stomach is dilated: the chlorides are often diminished and the phosphates increased; if the dilatation of the stomach be considerable, and the fermentation due to micro-organisms increased, the urine may contain an excess of the ethereal sulphates and of indican. The menstrual functions are, as a rule, not interfered with unless chlorosis be present or after profuse hæmatemesis. The occurrence of sugar is rare; albumin, on the other hand, is more frequently to be found.

Acetonuria and diaceturia in gastric ulcer.—It is now a well-established fact that acetone and aceto-acetic acid or diacetic acid are occasionally found in the urine in acute and chronic affections of the digestive tract, such as acute and chronic gastric catarrh, enteritis,

dilatation of the stomach, gastric ulcer, etc. Both bodies are often found together in the urine, as acetone is formed from the former; in some cases, however, acetone alone is found, and may be detected by the smell and the various reactions (41*a*, 56*b*), of which the nitro-prusside test is the handiest for clinical purposes, though not the most trustworthy ; whilst the presence of aceto-acetic acid is easily detected by the claret-red colour it gives with perchloride of iron. In many cases the presence of these bodies gives rise to no marked symptoms ; in others, however, we may find a group of symptoms which refer to the gastro-intestinal and nervous system, and which may assume a serious character resembling the toxic symptoms in diabetic coma. In the milder form we notice severe epigastric pain, persistent vomiting, headache, and giddiness. In the severe forms these symptoms may be associated with dyspnœa, drowsiness, coma, and epileptic convulsions. The symptoms usually set in suddenly and subside after a few days with the disappearance of the acetone from the urine. In most cases the affection is easily and early recognised by the peculiar acetone smell of the breath and of the urine. Lorenz (52*a*) noticed this affection in five cases of gastric ulcer, and I have observed diaceturia in three cases, in one of which the symptoms assumed an alarming aspect ; the patient, a girl aged twenty-two, who had had all the symptoms of gastric ulcer, was suddenly seized with violent abdominal pain, vomiting, and great prostration ; the vomiting persisted and could not be checked by any medicine, all food and even small quantities of water were rejected ; the patient had repeated attacks of syncope when she attempted to raise herself, the pulse was slow, and the temperature normal. The breath had a marked acetone smell ; the urine, which was scanty, had a sp. gr. 1028, smelled strongly of acetone, and gave the aceto-acetic acid reaction with perchloride of iron ; the symptoms persisted for five days, during which time the patient was fed by nutritive enemas ; citrate of potash in solution was given by the mouth and injected into the rectum. The symptoms then gradually subsided and the patient made a rapid recovery. It is held by many observers that a purely nitrogenous diet is often the cause of acetonuria (39*a*, 70*a*). This, however, is by no means the only cause, and of many cases of acetonuria, especially those occurring in diseases of the digestive tract, the exact causation is still obscure.

Nervous symptoms are common enough in gastric ulcer. Headache, vertigo, insomnia, and attacks of syncope are not uncommon ; nervous symptoms are often present also when neurasthenia, melancholia, or hysteria complicates the affection. The superficial reflexes vary ; in neurotic or hysterical subjects they are often increased, and in such cases the abdominal and epigastric reflexes may be exaggerated. Tetany, which has been known to occur on washing out the stomach, I saw in one case occurring spontaneously, and without lavage of the stomach.

The *temperature* is normal or even subnormal if the nutrition suffers much. Rise of temperature denotes some complication or some intercurrent disease.

The general nutrition in many cases remains good for some time; but with the duration of the disease it becomes much impaired, there is loss of flesh and the muscles feel flabby, the patient is pale, has an anxious appearance, the eyeballs are sunken, and the whole expression denotes much suffering. In younger persons the aspect is most commonly simply that of anæmia; in older persons it is cachectic rather.

The physical examination in many cases reveals nothing but the tenderness of the epigastrium and an accompanying cutaneous hyperæsthesia of that region; in very chronic cases, when the patient is much emaciated, a fibroid cicatrix may sometimes be felt as a band or as a slight intumescence, and in those rare cases in which a localised abscess has formed between the stomach and the adjacent parts (abdominal wall, spleen, liver, etc.), a distinct tumour, painful on percussion and pressure, may perhaps be felt. The discrimination of such an intumescence from the tumour of cancer is, as a rule, not difficult when the history and symptoms are carefully considered. Marked dilatation of the stomach can be ascertained by physical examination.

Alterations of the functions of the stomach in gastric ulcer.—*Hyperacidity.*—For some time great stress was laid on the increased amount of hydrochloric acid in cases of gastric ulcer, and no doubt free hydrochloric acid is found in the large majority of cases. In many cases the increased acidity is found soon after food is taken, and, at the height of the digestion, 0·3-0·35 per cent of hydrochloric acid may be found in the contents of the stomach. In some cases, even after digestion is completed and the food has left the stomach, there remains in the stomach a small quantity of fluid which contains hydrochloric acid (hypersecretion of hydrochloric acid, Reichmann); in these cases there is a prolonged secretion of hydrochloric acid, and these are probably the cases in which patients complain of pain in the epigastric region late at night or early in the morning when the stomach is empty. In about one-fourth of the cases of ulcer the quantity of hydrochloric acid is normal, in a few cases the acid is diminished (sub-acidity); this occurs in chronic cases when the ulcer is complicated by anæmia or chronic gastric catarrh, or when the stomach is dilated; though an excess of hydrochloric acid is sometimes noticed even in cases in which dilatation is distinct.

In many cases the hyperacidity can be made out by examination of the vomited matter; to use the stomach-tube in order to examine the gastric juice and the contents of the stomach after a test meal is only permissible when there has been no hæmatemesis; by the introduction of the stomach-tube we may produce not only hæmatemesis but, if the ulcer be acute and its floor thin, perforation also. The chemical tests for free hydrochloric acid are fully described in the article on "General Pathology of Digestion" (p. 281).

The secretion of *pepsin* does not undergo much alteration in gastric ulcer, occasionally it is increased; in old ulcers, where there is marked emaciation or dilatation of the stomach, it is often diminished.

The *motor* functions of the stomach are not interfered with by the ulcer unless there be marked dilatation, or some such complication as phthisis or chlorosis; though in a few cases the movements of the stomach are diminished (Korczynski and Jaworski).

Absorption from the stomach.—Zweifel gave iodide of potassium in gelatine capsules, and determined the length of time that elapsed before he could detect iodine in the saliva and in the urine; the absorption time was generally prolonged but very slightly, except in the early stages of ulcer when it may be more prolonged.

When the stomach is dilated we often find bacterial fermentation, sometimes with the formation of nitrogen, oxygen, carbonic acid, occasionally of hydrogen, sulphuretted hydrogen, and marsh gas.

Complications and Sequels of gastric ulcer.—*Dilatation of the stomach.*—The most marked cause of dilatation of the stomach in gastric ulcer is the narrowing of the pylorus due to the cicatrisation of an ulcer situated at or near the pylorus. This is a sequel rather than a complication.

Occasionally simple dilatation of the stomach without stenosis of the pylorus due to accompanying gastric catarrh or to muscular weakness may be present. As dilatation of the stomach is described in a separate chapter (p. 485) no further reference to its symptoms or treatment need be made here.

Hour-glass contraction of the stomach.—This will occur if an ulcer situated in the mid-region of the stomach undergo cicatrisation. It may give rise to the symptoms of dilatation, or to very indefinite symptoms which cannot be accurately interpreted. Inflation with air may sometimes reveal the peculiar outline of such a stomach. Jaworski points out also that in this condition there may be all the signs of dilatation, and large quantities of fluid may be introduced into the stomach, yet little can be drawn out by the syphon, as the fluid passes into the part beyond the constriction.

Perforation of the stomach is common, and is the most grave complication of gastric ulcer. It occurs much more frequently in acute ulcers or in an early stage of the chronic ulcer; it often causes the death of the patient by peritonitis; in rare cases a small perforation may heal, and the patient may completely recover; in other cases it may lead to the formation of an abscess, and in other cases, again, to the establishment of fistulous communication between the stomach and other organs.

Perforation into the general peritoneal cavity.—This, according to Welch, occurs in about 6½ per cent of all cases of gastric ulcer; it happens most frequently in young women. As already indicated this untoward complication is found chiefly in ulcers which are situated on the anterior wall of the stomach. Perforation may occur in cases in which there have been no definite symptoms of ulcer, or in which almost all symptoms have been absent and the patient apparently in good health. In other cases, however, perforation takes place after

local pain, vomiting, and even hæmatemesis have existed for some time. The symptoms of perforation are usually characteristic enough, unless death from collapse take place within a few hours after its occurrence.

The first symptom is sudden pain of severe and agonising character, which may be situated at first in the epigastrium, and then become diffused over the whole abdomen; or it may be seated rather over the right iliac fossa, and simulate the perforation of appendicitis or pyosalpinx; this I have seen in not a few cases (21). The pain may be continuous, or it may have the character of a severe colic. Along with the pain come the symptoms of collapse; the pulse becomes small, quick, and thready; the face is pale, and has a pinched, drawn appearance (*facies hippocratica*); the surface of the body is covered with cold, clammy perspiration; the voice becomes feeble, and the respirations frequent and superficial, and after a short time costal in character. The temperature, at first subnormal, becomes raised if the patient live for some time and peritonitis supervene. The tongue becomes dry; thirst is increased; vomiting is generally absent, if it occur at first it soon ceases, though often there is much retching; constipation is marked; the urinary secretion is diminished, and with the onset of peritonitis micturition becomes painful, or its secretion may be entirely suppressed; the urine may contain albumin and casts. On physical examination, besides the general features above noticed, we find the abdomen, as a rule, distended; but in a few cases, as pointed out by Wagner and myself, it may be retracted by the spasm of the abdominal walls; it is very sensitive to the touch, with local pain over the epigastrium or right iliac fossa. Percussion gives a tympanitic note all over the abdomen, with absence or considerable diminution of the liver dulness from the accumulation of gas between liver and diaphragm; this has been described as a pathognomonic sign of perforation, but it may be absent if adhesions exist between the stomach and liver; on the other hand, we may have this symptom without perforation in simple tympanites if coils of intestine distended with gas find their way between the diaphragm and the liver, and it may occur in simple non-perforative peritonitis. If much fluid pass out from the stomach the percussion note over the lateral and lower parts of the abdomen may become dull (a very rare occurrence); this symptom may also be noticed subsequently when peritonitis is well established. If the patient do not die from collapse during the first six to twelve hours, symptoms of peritonitis set in; the temperature may rise to 102° or 103°; the pulse becomes harder; the breathing becomes very shallow; retching and hiccough occur, and death follows from exhaustion or collapse. In some cases the pain becomes less severe, and the patient may seem better, yet death occurs in a short time.

Most cases of perforation from gastric ulcer end fatally: in a few cases, however, if the perforation be very small, and but little of the contents of the stomach effused into the peritoneal cavity, the opening

may be closed by deposits of lymph, and the patient may eventually recover. In a few cases, again, surgical interference, with the object of closing the opening into the stomach and clearing the abdominal cavity, has saved the patient from impending death.

Localised abscess the result of perforation.—In cases in which but a small quantity of the stomach contents has escaped, and in which before perforation adhesions, more or less firm, have been formed between the stomach and the neighbouring organs, we may have a local peritonitis with subsequent formation of an abscess. Whilst the symptoms at first are those of perforative peritonitis the further course varies according to the situation of the abscess; in many cases the general and local symptoms of peritonitis diminish; the abdomen is less distended and less painful, and the appetite improves; but strength is not regained, the pulse remains quick, the fever assumes a remittent type, and the patient eventually dies from exhaustion, or by way of pyæmia or of pylephlebitis. In other cases the patient improves considerably for a time till the abscess bursts into the peritoneal cavity, when he succumbs to general peritonitis; this is the case when the abscess forms behind the stomach, or between the stomach and the spleen, or is encysted in the mesentery; in rarer cases still the abscess after having perforated the diaphragm may open into one or other of the thoracic organs (pericardium, pleura, lung, mediastinum). In other cases, however, the pus from the abscess may, either directly or by a fistulous tract, be discharged outside or become accessible to operative measures, and the patient recover.

Occasionally the abscess is situated anterior to or above the stomach, where it forms a distinct swelling painful to palpation, and dull on percussion, and where the pus can be easily reached by puncture or incision (21); however, usually the abscess is not easily accessible to the surgeon's knife. One form of localised abscess that results from perforation of a gastric or duodenal ulcer, or occasionally from appendicitis, gall-stones, perinephritic abscess, or cancer of the stomach, presents peculiar clinical features, and requires a fuller notice, namely, the *subphrenic abscess* or pyopneumothorax subphrenicus (Leyden). This malady is fully described in a later chapter (p. 570).

Perforation of a gastric ulcer, without the intervention of an abscess, may take place either into a hollow or solid organ to which the stomach by new-formed inflammatory tissue has previously become united. If perforation take place into a part of the intestine, we get the establishment of a *bimucous fistula* (21); the communication may be made between the stomach and the duodenum, or any other portion of the intestine; most commonly it is made directly with the transverse colon (gastro-colic fistula), and the condition may be diagnosed by the rapid passage of food through the digestive tract.

If adhesion take place between the stomach and diaphragm the ulcer may perforate into the thoracic cavity, and thus *pyopneumothorax* or *gangrene of the lung* may be established. In rare cases the pericardium

has been perforated, with the result of pneumo-pericardium and gangrenous pericarditis. Debove and Renault give a brief description of some of the recorded cases. A sudden epigastric pain is followed by dyspnœa and cyanosis; the apex beat of the heart cannot be felt, the cardiac dulness is replaced by a tympanitic note, pericardial friction sound may be heard, the internal heart sounds may be accompanied by a loud metallic timbre, the pulse becomes rapid and feeble, and the patient dies within a short period after the occurrence of the perforation. In yet rarer cases the pericardium may become adherent to the heart, and the ulcer of the stomach, by an extension of the ulcerative process, may burst into the heart. According to Debove and Renault only four such cases are on record; the prominent symptoms are rapidly fatal hæmatemesis with melæna. One case is recorded by Dr. Finny.

If the perforation take place into the mediastinum—an extremely rare occurrence—interstitial emphysema may arise; and, if death do not follow quickly, mediastinal abscess may follow.

Another complication of gastric ulcer is the supervention of *cancer*. This coincidence was already known to Dietrich and Brinton, who drew especial attention to the subject; according to Lebert 9 per cent of all cases of cancer of the stomach owe their origin to simple ulcer of the stomach, but according to Rosenheim only 6 per cent; Haeberlin, again, reduces the number to 2-3 per cent. The observations of Hauser have thrown some light on the implantation of cancer on the ulcer; for he noticed masses of epithelial cells amidst the fibrous tissue at the borders of the ulcer, from which cells cancer may take its origin. The naked eye and histological appearance of the cancer thus formed does not differ from other cancers found in the stomach; but the clinical features of such cancers, as a rule, are for a long time those of gastric ulcer, till eventually a tumour appears, and with it cancerous cachexia; yet even then the gastric juice still shows the presence of hydrochloric acid, and sometimes even of hyperacidity. In other cases the history of a case of cancer with the ordinary symptoms indicates that symptoms of an ulcer had existed years before (21). [*Vide* "Tumours of Stomach," p. 555.]

Course and duration of gastric ulcer.—From the symptoms of gastric ulcer given above, it is evident that the disease shows an extreme diversity both in its features and in its subsequent course and terminations. Anatomically we distinguish between acute and chronic ulcers, and clinically the same classification has been adopted; but, as gastric ulcer may exist a long time without producing any symptoms, we must not assume that, if hæmorrhage from the stomach or sudden perforative peritonitis occur in a person presumably healthy, we have to do with an acute ulcer; it is more likely that the ulcer has existed for some time in a latent state. Many cases of gastric ulcer (according to Cruveilhier about 80 per cent) end in recovery; in many we have remissions which may occur at intervals of months or years; and cases are not infrequent in which ten and twenty years after the occurrence of the first symptoms the characteristic signs of gastric

ulcer are noticed again; no doubt in these cases the old ulcer had cicatrised, and either had broken out afresh or given place to a new ulcer: in other cases, some time after the disappearance of the characteristic symptoms the signs of dilatation of the stomach appear; in others again, as stated above, either cancer supervenes or one of the other complications occurs.

Clinically the following forms may be distinguished:—

(i.) *Acute hæmorrhagic form.*—Either without previous symptoms or after symptoms of very short duration profuse hæmatemesis occurs. The case may end fatally, or symptoms of chronic ulcer may supervene; or, again, in a few cases, no further symptoms may appear.

(ii.) *Acute perforating ulcer.*—Either without any previous symptoms or after slight gastric disturbance, sudden perforation into the abdominal cavity occurs, in a very large majority of cases with a fatal termination.

(iii.) *Chronic dyspeptic form.*—In this there are marked dyspeptic symptoms, with moderate pain and occasional vomiting.

(iv.) *Chronic gastralgic form.*—Gastric pains predominate; vomiting may or may not occur.

(v.) *Chronic hæmorrhagic form.*—This resembles the chronic dyspeptic form, with hæmatemesis superadded.

(vi.) Some authors speak also of a *cachectic form*, in protracted cases, of which emaciation, anæmia, and a cachectic appearance—not unlike that seen in cancer—appear; of (vii.) a *stenotic* form, with symptoms of dilatation of the stomach; and of (viii.) a *recurrent* form in which, long after apparent healing of the ulcer, the symptoms recur.

Diagnosis.—In many cases of gastric ulcer the diagnosis is easy; in others the diagnosis can only be arrived at after a careful analysis of all the symptoms, and after watching the patient for some time; whilst in not a few cases a definite diagnosis is impossible, as most of the symptoms are absent. None of the symptoms mentioned above is absolutely characteristic of gastric ulcer, and all may occur in other affections of the stomach and in other diseases.

The chief diseases from which gastric ulcer must be distinguished are:—

(*a*) Chlorosis with marked dyspeptic symptoms, such as loss of appetite, pain after food, and occasional vomiting. The absence of localised pain, the irregularity of the vomiting and its independence of meals, will often enable us to distinguish this condition—due to anæmia and hyperæsthesia of the stomach—from gastric ulcer; on the other hand, as gastric ulcer often occurs in chlorotic women, it will be well, if in doubt, to treat the case as one of gastric ulcer, and to notice the effect of the treatment: if the symptoms be due to gastric ulcer the appropriate treatment may give relief in a few days; if, on the other hand, they are due to chlorosis, a good effect should follow the administration of iron.

(*b*) *Gastric catarrh.*—Acute catarrh can scarcely be confounded with gastric ulcer. In subacute and chronic gastric catarrh we have the following distinguishing features: the pain is not localised; it is not so severe as in ulcer; it is not increased by pressure; flatulence, distension,

or discomfort for hours after food generally accompanies it; vomiting occurs at irregular intervals; the vomit contains mucus, and there is no hyperacidity of the gastric juice; hæmatemesis is extremely rare, and if it occur the quantities of blood are small.

(*c*) *Nervous dyspepsia.*—In this condition we occasionally meet with cases in which the differential diagnosis is a matter of difficulty and sometimes of impossibility. The examination of the gastric juice does not help us much; in some of these cases there is hyperacidity, often indeed a continuous secretion of hydrochloric acid (the so-called Reichmann's disease); the epigastric pain may be very severe (gastralgia); it is usually sharp, comes on when the stomach is empty, is relieved by food, and is not increased by pressure. Nervous dyspepsia includes a number of different affections which are difficult to classify.

(*d*) *Cancer of the stomach.*—The most important points to bear in mind here are: that cancer occurs in most cases after the fortieth year; the pain is more continuous and not so dependent on food; the appetite soon fails; vomiting occurs later in the disease, and has rather the character of the vomit of dilatation of the stomach; hæmatemesis is not so profuse, and has the coffee-ground character. The presence of a tumour in or near the epigastric region is the most characteristic symptom of cancer, yet it must be remembered that in some cases of cancer no tumour can be felt; on the other hand, in ulcer of the stomach, as already stated, a fibroid cicatrix or a local abscess may simulate a tumour. Equally important in cancer are the usual absence of hydrochloric acid in the vomit and in the gastric juice, the steady loss of weight, and the cachectic appearance; we must not forget, however, that ulcer in older subjects may be attended by more or less cachexia, whilst in some cases of cancer, especially if situated at the cardiac end of the stomach, or diffusely infiltrated, the appearance of the patient is more anæmic than cachectic; and, again, the subjects of cancer under appropriate treatment may show a distinct increase in weight for a time.

(*e*) *Ulcer of the duodenum.*—The differential diagnosis can only be made in a few cases in which there is melæna, and a deeper pain to the right of the epigastrium occurring several hours after food; or in which, with symptoms of gastric ulcer, the vomit has an alkaline reaction, contains bile, and digests fibrine (Boas). It occurs more frequently in men of middle age.

(*f*) *Gall-stones.*—In the majority of cases the distinction between biliary colic and the gastralgia of gastric ulcer is easy; but we meet with cases of gall-stones in which the main symptom is at first a severe local pain with or without vomiting, and in which jaundice or the definite symptoms of biliary colic are deferred. The chief points of distinction are: (*a*) As regards the pain; in hepatic colic the pain comes on suddenly, some hours after a meal or at night; especially after an indiscretion of diet, a muscular effort, or mental shock, or excitement. The pain is very severe, and is rather colicky than burning in character as in gastric ulcer; it is not situated in the epigastric region (except when the

gall-stone obstructs the common duct near its termination, in which case jaundice is invariably present); it lasts for many hours and subsides suddenly; it is associated with a pain over the right shoulder-blade, and on the subsidence of the pain or soon after, the epigastric region often ceases to be tender to touch or pressure; at times, however, the pain on pressure persists. (β) As regards vomiting, this occurs soon after the beginning of the attack, and persists for some time; at first every kind of food is vomited very soon after it is taken; the vomited matter shows no hyperacidity. (γ) A rise of temperature with or without a shiver is characteristic of gall-stones. (δ) Profuse sweating and great prostration likewise speak for gall-stones. (ϵ) The conjunctivæ show at times a distinctly icteric tint, and the urine may contain small traces of biliary colouring matter even though there be no jaundice apparent.

(*g*) *A movable or displaced kidney* may produce severe gastralgic pains and vomiting, and so simulate a gastric ulcer. A bimanual examination will, as a rule, clear up the diagnosis. Usually symptoms of neurasthenia are present in these cases.

(*h*) The diagnosis of complications has already been spoken of under symptomatology; for the distinction of dilatation of the stomach due to ulcer from dilatation due to other causes I must refer to the article on "Dilatation of the Stomach" (p. 485).

Diagnosis of the seat of the ulcer.—This in many cases is not possible, and no great reliance can be placed on the situation of the pain: it is said that an ulcer on the posterior surface gives rise to pain situated in the back rather than in the epigastrium, and is increased by the recumbent posture; here also it is said to give rise more often to hæmorrhage than if situated on the anterior surface; in the latter situation it is said to give rise to pain to the right or to the left of the middle line in the epigastrium—pain which is lessened when the patient lies on the left side. Perforation into the abdominal cavity is more common with ulcer on the anterior surface; ulcer at the pylorus is said to cause pain in the middle line or to the right of the middle line, and the pain does not occur till some time after the ingestion of food; cicatrisation of the ulcer in this situation causes dilatation of the stomach.

The prognosis of gastric ulcer is, on the whole, favourable. Many patients completely recover, especially if the ulcer does not extend to the serous coat, and does not persist too long. The cicatrisation of a chronic ulcer may give rise to stenosis of the pylorus with dilatation of the stomach, and to adhesion between the stomach and neighbouring organs, events which in their turn may give rise to a series of fresh symptoms and troubles. Again, relapses are not uncommon. According to Lebert the mortality of gastric ulcer is about 10 per cent; about 6½ per cent terminate fatally by perforation into the peritoneal cavity; death from hæmorrhage is said to occur in about 3 per cent; in a few death is due to exhaustion or to one of the rarer complications mentioned above.

Treatment. — *Prophylaxis.* — We have seen that gastric ulcer frequently occurs in young chlorotic women. It is well, therefore, in all cases of chlorosis to regulate the diet carefully, and to see that the patient avoids very hot food, coarse food, and food rich in acid and condiments which irritate the mucous membrane of the stomach. Continued pressure on the stomach, such as is produced by tight lacing or by a faulty stooping posture, is also to be avoided (61).

The treatment of the ulcer will vary with the stage of the disease, and the symptoms that may be presented: the objects to be fulfilled are the healing of the ulcer, the avoidance of all irritating agents and the relief of troublesome symptoms. This end is best attained by rest, not only of the stomach, but also of the body generally. This treatment, known as the treatment by rest (rest cure), first recommended by English physicians, and lately by Leube, is to be carried out in a systematic manner. Rest of the body is best effected by the recumbent posture: rest of the stomach by feeding the patient entirely by nutritive enemas; but as this latter is only possible for a short time, food when administered by the mouth should be easily digestible and not irritating. This dietetic treatment is further helped by the administration of alkalies to diminish the hyperacidity of the gastric juice, and by the application of hot poultices to the epigastric region.

The course of treatment in by far the largest number of cases resolves itself into this: the patient will remain in bed for some weeks, in fact till the chief symptoms have subsided; hot poultices are to be applied to the epigastric region or, instead of poultices, the stomach capsule first introduced by Leube. This consists of a tin capsule filled with hot water and so shaped as closely to fit the epigastric region; it has a metallic ring on each side, so that it can be easily fastened, and so may be worn even when the patient is about.

To give the stomach complete rest some have recommended feeding by enema exclusively (20, 87), but it is scarcely advisable to treat all cases in this manner without distinction; nor is it possible to carry out this treatment for a long time without causing the evils of inanition. Exclusive rectal feeding is indicated when the vomiting of food is persistent; when the ingestion of food causes great pain; when there is hæmorrhage from the stomach, and when perforation has occurred, or appears to be threatening. But even in these cases exclusive rectal feeding rarely need be prolonged beyond a week or a fortnight. The patient takes nothing per mouth except small pieces of ice or small quantities of water to quench the thirst; for the rectal feeding many physicians recommend artificially digested food, such as peptonised milk-gruel, peptone suppositories, and the like: many, however, prefer to give enemas of beef tea and raw eggs, with a little brandy (in all about two to three ounces); the peptonised food is more readily absorbed, but the observation of Ewald shows that enemas of eggs act quite as well and are quite as well absorbed; peptonised suppositories sometimes irritate the bowels, or are not well retained, and, unless recently prepared, are sometimes passed out again unaltered.

Before giving the nutritive injection the rectum should be emptied by one of water; the nutritive enema may be given every four to six hours. Milk-gruel is prepared by mixing equal parts (about ten ounces) of milk and thick gruel, and to this two teaspoonfuls of Benger's liquor pancreaticus and thirty grains of bicarbonate of soda are added. To increase the nutritive value an egg may be added to the milk before the addition of the liquor pancreaticus. Peptone suppositories weigh about sixty grains, and contain between 40 and 60 per cent of peptones.

Milk occupies the first place in the dietary; it is alkaline, it lessens the acidity of the gastric juice; it does not irritate the mucous membrane, either mechanically or chemically; it does not call forth vigorous peristaltic movements of the stomach, and it does not remain long in the stomach. Not more than four to six ounces should be taken at once, and in twenty-four hours three to four pints may be taken. The milk, which ought to have been well boiled, may be taken warm or cold; when it produces acidity, alkalies, such as carbonate of soda, may be added to the milk; when it is not well borne peptonised milk should be tried instead. In some cases milk in any form cannot be tolerated, and if so, besides rectal feeding, we should try freshly-expressed beef juice, weak beef tea, Liebig's beef extract, or some of the prepared beef preparations, such as Brand's essences or Valentine's beef juice. In some cases of vomiting, which persisted even in patients fed by nutritive injections, scraped raw beef, taken at first in very small quantities, has been well borne, has given relief to the pain and checked the vomiting. I have often, therefore, in cases of gastric ulcer given the scraped raw beef early in the course of the case with considerable benefit.

If the patient get tired of milk, or if it be not well borne, Benger's food, Nestlé's food, powdered rice or arrowroot, raw egg, and the above-mentioned beef extracts may be tried.

An exclusive milk diet should be continued for three to four weeks. The epigastric pain, as a rule, has disappeared, and the vomiting has stopped long before this time; but it is well to continue the treatment so as to give the stomach rest and avoid irritation: then, for a week or so, the patient may still have liquid diet, but with the addition of biscuit or stale bread. He may take bread and milk or milk boiled with a small quantity of sifted flour; he may also take arrowroot and tapioca. After a week of this diet, if the pain do not return, the patient may pass on to solids, such as boiled fish, chicken, pigeon, sweetbread or tripe; scraped beef and raw eggs may also be given with this dietary. Stimulants had better be avoided altogether, unless perhaps small quantities of claret or light Burgundy be allowed. With this diet, which may be continued for some weeks, the patient often gains weight; and he may be allowed to take gentle exercise. Gradually mutton chops and small quantities of underdone steak may be taken with stale bread. Vegetables, however, with the exception of rice, are still to be forbidden, as are cakes and pastry. Milk and small rice puddings may be allowed. Alcoholic stimulants are better avoided. The duration of this dietary depends on

the condition of the patient: if he feel sufficiently strong he may now follow his occupation, and make gradual additions to his dietary; the more indigestible food-stuffs, such as vegetables and pastry, should, however, not be taken at any time. Many are the diet sheets given by various observers, and adopted in various institutions and private hospitals where patients with gastric ulcer are treated (61).

Patients who have suffered from gastric ulcer should be careful in diet, not only for weeks, but for months, and with the return of any such symptoms as pain or vomiting the treatment with rest and strict diet should again be enforced.

For the treatment of chronic ulcers in which there is marked dilatation of the stomach I refer to the article on "Dilatation of the Stomach" in this volume.

Almost as essential as the diet is the strict supervision of the function of the bowels. Constipation is a common accompaniment of the gastric ulcer, and its proper treatment is as important as the dietetic measures. Carlsbad salts, or a saline aperient such as a combination of sulphate of soda and sulphate of magnesia, or some mineral water, such as Carlsbad water, Hunyadi Janos, or Æsculap, are the chief aperients recommended. Carlsbad salts enjoy special favour with Continental observers—one or two teaspoonfuls are given in a tumblerful or half a tumblerful of warm water before breakfast. These salts, apart from their purely aperient effect, are said to act beneficially by diminishing the acidity of the gastric juice, by stimulating the secretion, and by increasing the peristalsis of the stomach. In many cases the good effects of this aperient cannot be doubted; but in some the salts increase the pain and discomfort after meals, and diminish the appetite: in such cases I have found simple enemas and the administration of an infusion of senna pods to be of service. Eight to ten senna pods are added to ten ounces of boiling water, the water is allowed to cool and to stand for some hours, and then the pods are removed. Generally after the infusion is taken a satisfactory action of the bowels follows in six to eight hours. Rhubarb and cascara sagrada are useful in the more chronic cases.

Medicinal treatment in many cases may help the dietetic means. The remedies which are here to be specially recommended are bismuth in the form of subnitrate or carbonate, and bicarbonate of soda. Bismuth salts have long been recommended by English writers: the salt may be given either in powder or in suspension; the doses usually given are from ten to fifteen grains. Recently, however, much larger doses of bismuth have been recommended by Kussmaul (28). One hundred and fifty to three hundred grains of bismuth are suspended in about eight ounces of water, and the mixture is passed into the stomach, previously washed out by means of the stomach-tube: it is then allowed, by guiding the tube, to remain in the stomach for fifteen minutes, during which time the patient occupies such a position that the bismuth is brought into contact with the ulcerated surface (if the situation of the ulcer can be determined); after this lapse of time the fluid is allowed to run out again

through the tube. At first this method is applied daily; after a time, once in two or three days. When the passage of a stomach-tube is contra-indicated the bismuth mixture is to be drunk. The bismuth is supposed to act mechanically by forming a covering which protects the ulcer and facilitates the healing of it.

This treatment, according to Matthes (56), Fischer, Steitzing, and others, gives most satisfactory results, and appears to bring about a much more speedy healing of the ulcer. Against the method several objections have been urged, such as the danger of using the stomach-tube, or the evil effects of large doses of bismuth which may set up vomiting and diarrhœa. For some years I have given very large doses of bismuth in cases of gastric ulcer—twenty to fifty grains three or four times daily: I have never seen any ill effect from these large doses; on the contrary, in most cases the large doses, especially if a small dose of morphine or hydrochlorate of cocaine were added, relieved the epigastric pain very speedily and also quickly stopped the vomiting. I agree with other observers, such as Mathieu, Savlief, Wegele, in thinking that the administration of large doses of bismuth are so well borne and give such good results that one may generally dispense with the irrigation method.

Of the various preparations of bismuth the subnitrate appears to answer best, and it can be given either in powder or in suspension; ten to fifteen grains should be given at first, and the dose may afterwards be increased to twenty and thirty grains or more: the bismuth should be given before meals, and if the pain be very great, or the irritability of the stomach excessive, it may be combined with one-tenth of a grain of morphine or one-fourth grain of hydrochlorate of cocaine; if the appetite be deficient, small doses of hydrocyanic acid and tincture of nux vomica may be added to the mixture.

The carbonates of soda, lime, and magnesia have for a long time been given in cases of gastric ulcer. Of these the first—the bicarbonate of soda—is still largely given; some French observers, notably Debove, recommend very large doses (300 to 450 grains) per day; they report very good results, and state that such large doses are very well borne by the patients, who complain only of increased thirst and increased urination; occasionally, however, such large doses produce rather profuse diarrhœa, which can, however, be obviated by giving some carbonate of lime with the bicarbonate of soda.

Nitrate of silver is also an old remedy for gastric ulcer, and I have been in the habit of giving it for years, especially to patients, such as the out-patients of hospitals, who cannot afford to undergo the rest treatment; I have found in many cases that it relieved the pain even better than morphia, and that it gave relief to the other symptoms also. I have given the nitrate of silver either in the form of pills (one-fourth of a grain to the dose) or in solution (one-fourth of a grain). Boas recommends it very highly in the liquid form, and gives it in small but gradually increasing doses, beginning with half a grain three times daily and going on gradually to one grain. I have given the nitrate of

silver in more than 200 cases, and have never seen any signs of argyria follow its administration; it may be given for several weeks and then discontinued for some time.

Treatment of symptoms and complications.—Pain.—The application of hot poultices and rest, and regulation of the diet, often suffice to relieve the pain; the use of bismuth and nitrate of silver has also been mentioned for the severe paroxysms of pain (gastralgia). Codeia, or better still morphine, either by the mouth or subcutaneously, is indicated: given in the latter form, morphine, according to Leubuscher and Schäfer, and Hitzig, diminishes the secretion of hydrochloric acid in the gastric juice.

Vomiting.—For persistent vomiting abstention from food by the mouth for some days is to be recommended; of drugs drop doses of hydrocyanic acid, or powders of bismuth with cocaine, often act well.

Hæmatemesis.—The patient is to be kept absolutely at rest and in the recumbent posture (he should not even be allowed to get up to pass urine or fæces), and all food by the mouth should be avoided; if the patient be excessively thirsty he may swallow small quantities of ice, but even this should be withheld if possible. If the hæmorrhage persist, ergot in the form of ergotin should be injected subcutaneously and ice applied to the epigastrium. In profuse hæmatemesis I have given turpentine with the greatest benefit; it may be administered either in the form of capsules or as an emulsion (two to three teaspoonfuls of turpentine beaten up with the white of one egg). The dose, about twenty to thirty minims, may be repeated after some hours if the hæmorrhage persist. I could cite several cases of hæmatemesis from ulcer in which the patient was pulseless and blanched to an extreme degree, in which ice, gallic acid, ergotin injections, and other styptics had been tried in vain, and in which the first dose of turpentine completely stopped the hæmorrhage. It is well borne, and I cannot speak too highly of its immediate and beneficial action.

If the anæmia produced by the hæmorrhage be excessive (the pulse scarcely perceptible, the patient showing signs of syncope, and the voice becoming feeble), then transfusion or injection into the subcutaneous tissue of one pint or more of normal salt solution may be tried.

For some days (three to six) after the last hæmorrhage the patient must be fed exclusively by enema, and must keep the recumbent posture; after that period the treatment for the healing of the ulcer by rest, liquid food, and hot applications to the epigastrium must be systematically carried out. In cases in which profuse hæmorrhages occur from time to time and exhaust the patient, an operation may be advisable. This was successfully done in one case by Kuster, who cauterised the ulcer and performed gastro-enterostomy.

Treatment of perforation of the ulcer.—From the beginning of the symptoms the patient is to be kept absolutely quiet, all food by the mouth is to be stopped, large doses of opium are to be given, and the patient is to be kept under its influence; when opium is not well borne,

morphia may be given subcutaneously; or opium or codeia in suppositories, or in small enemas. At the same time, as a stimulant citrate of caffeine may be given by the mouth or subcutaneously, and rectal injections of brandy.

The treatment of the perforation by *surgical means* now requires a few words of consideration. In some cases the patient is so collapsed soon after the event that no operation is likely to be of any benefit; indeed the patient may die from shock: in a few cases the symptoms of collapse pass off and the patient recovers without operation; many cases, however, remain in which an operation is feasible.

Considering the high mortality of cases of perforation, which may be estimated at about 95 per cent, the results obtained by English surgeons by operation are certainly very encouraging. Jowers tabulated 25 cases, of which 8 recovered; Barling collected 37 cases with 13 recoveries; and since then several other successful cases have been recorded. It is difficult to give the indications for operative interference; it would seem well to wait till the shock caused by the perforation has passed off, and to act before symptoms of collapse have set in; but the moment is not easily determined. Many of the patients in whom recovery has taken place were operated on when in a state of collapse, with small, thready pulse, profuse clammy perspiration and subnormal temperature; hence it is now widely held that as soon as perforation has been diagnosed, the abdominal cavity should be opened, the perforation looked for, and closed by sutures (excision of the ulcer is rarely possible), and the peritoneal cavity cleansed and drained. The cases in which perforation occurs when the stomach is empty, so that no great extravasation of food into the abdominal cavity takes place, and perhaps those also in which the edges of the ulcer are not much indurated, present a better prognosis than those in which perforation occurs soon after a full meal. On the subject of the surgical treatment of gastric ulcer I must refer to the treatises on surgery of Gould, Heineke, Maylard (56*a*), and Comte. For the treatment, both medical and surgical, of the symptoms produced by stenosis of the pylorus, giving rise to dilatation of the stomach, and of those due to the formation of a subphrenic abscess, I refer to the respective articles on these subjects in the present volume. Surgical treatment has also been recommended in cases of long-standing gastric ulcers, where the pain persists and the patient becomes weak and unable to follow his work. In these cases gastro-enterostomy has sometimes given excellent results, as in the cases operated on by Doyen.

Ulcer of the Duodenum

An ulcer of the same character as the gastric ulcer is found in the duodenum occasionally. The symptoms vary somewhat with its exact position. It is of these ulcers only that we can treat here. Ulcerations of a tuberculous nature, ulcers in enteric fever, and cancerous

ulcers, which are occasionally found in the duodenum, are not included in this article. For the earlier bibliography the reader is referred to Bucquoy.

Ulcer of the duodenum is of much rarer occurrence than gastric ulcer.

Etiology.—*Sex.*—According to most observers it occurs more frequently in men than in women. It is stated that it is 7-8 times more frequent in man than in woman; our own statistics on a small number of cases give a high proportion of men, and according to the analysis of Drs. Perry and Shaw—who analysed the post-mortem records from 17,652 autopsies at Guy's Hospital, from 1826 to 1893—of 56 cases of perforating ulcer of the duodenum, 14 occurred in women and 42 in men: if burns are excluded the rate is 8 females to 42 males.

Age.—Duodenal ulcer may occur at any age; Sir Andrew Clark noted it in a child 10 years old. Several cases are recorded in persons over 70 years of age; the average age is stated to be between 35 and 40. Ulcers due to burns occur much more frequently in children.

Occupation and habits.—On this subject we have no trustworthy data. Alcohol is regarded by some as an etiological factor—with very doubtful truth, however. Of 10 fatal cases of duodenal ulcers recorded in the post-mortem books of the Manchester Royal Infirmary only one was in a drunkard.

Tuberculosis.—Some have drawn attention to the rarity of duodenal ulcers in tuberculous subjects. Perry and Shaw collected 25 cases of ulceration of the duodenum in association with tuberculosis; of these, 11 only appeared to be of tuberculous origin. These were associated with extensive tuberculous ulceration of the rest of the alimentary tract, and the duodenal ulcers were small, thickened at the edges, and shallow; the other 14 cases had more of the character of gastric ulcers.

Among other diseases in which duodenal ulcers have been found may be mentioned Bright's disease, heart disease, septicæmia, and enteric fever.

Burns.—Curling first drew attention to the occurrence of duodenal ulcers after extensive burns of the skin; and he was able to collect no less than 10 cases. Since then the subject has been investigated by several observers, especially in England, with the result that, though the occasional appearance of an ulcer after an extensive burn has now been well established, the occurrence is certainly very rare. Mr. Holmes examined the record of 125 fatal cases of burns and found duodenal ulcer sixteen times. Perry and Shaw noticed it five times in 149 autopsies of cases of burns. It is further established that these ulcerations occur chiefly in young subjects, and more especially when there are extensive burns of the trunk; that the ulcer occurs in the second or inflammatory period of the burn; that before ulceration occurs there is marked congestion followed by hæmorrhagic infiltration which affects other parts of the alimentary tract besides the duodenum. In several cases (one noted by myself in which death took place six days after

extensive burns) the lesion did not go beyond a hæmorrhagic infiltration; in other cases an ulcer forms which may lead to perforation or to profuse hæmatemesis. The formation of the hæmorrhagic infiltration is most rationally explained by a septic embolus, and the change into an ulcer by the action of the gastric juice.

Dr. W. Hunter succeeded in producing extensive ecchymoses and ulcers of the duodenum in dogs by injecting toluylendiamin, and suggested, as a probable explanation of the duodenal ulcer in burns, the secretion, with the bile, of a poison which causes the ecchymoses and ulceration. Experiments by Ponfick and others have shown that in animals extensive scalds will cause a destruction of the red blood corpuscles similar to that seen on injecting toluylendiamin. Considering, however, that after burns ecchymoses are found in other parts of the intestinal tract as well as in the duodenum, Dr. Hunter's view can scarcely be regarded as well established. Moreover, Dr. Fenwick found in dogs that ulceration of the duodenum occurred after toluylendiamin poisoning after ligature of the common bile-duct.

Pathological anatomy.—*Situation.*—In most cases the ulcer is situated in the first part of the duodenum, and here most frequently close to the pyloric ring; in rare instances it has been found in the second, and more rarely still in the third part of the duodenum (out of 149 cases it was found 123 times in the first part; 16 in the second part; twice in the third part: in one of these last the ulcer was probably tuberculous). The ulcer is found most frequently on the anterior wall; sometimes on the upper wall.

Number.—Usually one ulcer only is found; occasionally there may be two or more, and if so they are generally found in different portions of the duodenum.

In *form* and *dimension* they resemble the gastric ulcer; they are generally circular, but may be oval or elliptical; those situated near the pylorus are generally larger, but this rule does not hold good invariably.

Appearance.—The ulcer has the same appearance as the gastric ulcer, it has the punched-out character; but as it is generally chronic the edges of the ulcer are thickened. The floor is formed by the submucous or muscular coat, and in some chance cases by the liver, pancreas, or gall-bladder, as adhesions between the duodenum and the neighbouring organs may arise. Occasionally, as in a case recently seen, the neighbouring portion of the mucous membrane shows polypoid excrescences.

The microscopic examination of the ulcer shows the same inflammatory changes at the borders as is the case in the gastric ulcer.

Duodenal ulcer may give rise to pathological consequences similar to those of ulcer of the stomach; hæmorrhage and perforation are the most common, subphrenic abscess has been repeatedly noted, cicatrisation may give rise to stenosis of the duodenum with subsequent dilatation of the stomach, or, if the ulcer be situated over the biliary papilla, to

persistent jaundice. The ulcer may perforate into the gall-bladder (Eichhorst), or into another part of the intestines, causing a bimucous fistula; or it may perforate the aorta (Stich), or cause a thrombosis of the portal vein (Frerichs). It may give rise to a cancerous growth.

Pathogeny.—The character of duodenal ulcer is the same as that of ulcer of the stomach, and its formation is due to the same cause or causes. That the gastric juice plays an important part in its formation is shown by the occurrence of the ulcer—in by far the majority of cases—in the first part of the duodenum, where the acidity of the gastric juice is not as yet neutralised by the alkaline pancreatic juice. It is highly probable also that a disturbance in the circulation, leading to a localised hæmorrhagic infiltration similar to that observed in ulcers of the stomach, occurs as the early stage; and probably the same causes operate here which were duly considered in the pathology of gastric ulcer: bacterial infection probably plays an important part in the formation of the ulcer.

Symptomatology.—Anatomically we may distinguish the acute and chronic ulcers of the duodenum, but clinically this distinction can scarcely be made with any certainty; for though the first characteristic symptoms may be acute, such as profuse hæmorrhage or signs of perforation, yet the ulcer may have existed for a long time previously without having given rise to any definite symptom. Leaving this distinction out of sight, we may divide cases of duodenal ulcer from the clinical point of view into certain groups, namely, those in which the course is chronic and in which symptoms arise which enable us to diagnose the duodenal ulcer; those in which the first symptoms are due either to excessive hæmorrhage (melæna with or without hæmatemesis) or to perforation; and those in which signs of stenosis of the duodenum or of the common bile-duct form the prominent feature. In many cases, however, the symptoms are so indefinite that no diagnosis can be made.

Kraus estimates the number of latent cases as about one-fifth of all cases observed. Ulcers situated close to the pylorus are not to be distinguished from gastric ulcers, though it is asserted that in these cases the localised pain is situated more deeply and to the right of the epigastric region, and that melæna occurs before the hæmatemesis; but these signs may be present in gastric ulcer, as we have already seen.

Considering the cases of our first group, the chief symptoms which indicate the diagnosis of duodenal ulcer are deep-seated and localised pain occurring several hours after food, dyspeptic symptoms, and melæna.

The pain varies in intensity: sometimes it is of a burning, lancinating character; at other times the patient only complains of a painful distension. The pain is situated in the right hypochondrium between the liver and the umbilicus; it radiates thence to the epigastrium, right shoulder, or umbilicus (the umbilical pain is most likely the associated pain in affection of the duodenum, but as it is also associated with affection of other portions of the small intestine, it is of no great

diagnostic importance); it is said not to be accompanied by pain over the dorsal vertebræ; it is generally not a continuous pain, but occurs several hours after meals, and lasts for some time; it is increased by pressure. According to Chvostek the time of its appearance after food varies with the quantity and quality of the food; it occurs earlier after breakfast, later after a more copious meal. *Melæna* may be so slight as not to be perceived by the patient or profuse, occurring only once, or several times in a short space of time. The patient may feel a colicky pain, and a desire to go to stool immediately after; or he may have a sudden feeling of faintness, accompanied by vomiting and loss of consciousness; or, in very severe cases (of which I saw one example in which the diagnosis was verified by autopsy), he may have epileptic convulsions. As the patient is himself not always conscious of having passed blood, and as sometimes the discharge of blood may not occur till some time after the above symptoms have appeared, it is always well—as already mentioned when speaking of gastric ulcer—in cases of sudden faintness, marked by anæmia, in which dyspeptic symptoms have existed for some time, to examine the stools. The *dyspeptic symptoms* differ somewhat; often the appetite is not impaired, but the patient complains of a fulness and heaviness coming on some hours after a meal; he often also complains of acidity and water-brash; vomiting may be altogether absent, or occur at long intervals; there is more often diarrhœa than constipation. The health of the patient suffers at first but little; after a time there is emaciation. If the attacks of melæna occur often, or if a large quantity of blood be discharged, the patient becomes very anæmic and weak.

In the second group of cases, the first and chief symptoms for which we are called upon to treat the patient are either those of profuse hæmorrhage from the bowels or of perforation. If hæmorrhage in the form of melæna be the first symptom, we have to inquire into the history and the presence of localised pain in the abdomen, as melæna may occur in other affections such as cirrhosis of the liver, cancer of the intestines, chronic valvular affection of the heart, dysentery, pernicious anæmia, purpura, and so forth. It is not until we have eliminated these affections that the diagnosis of duodenal ulcer can be made with some degree of certainty.

The symptoms are those of any internal hæmorrhage, and need not be described again. In some cases the melæna disappears after a few days; the patient remains anæmic and weak for some time and then gradually recovers; in other cases the patient may die from syncope or collapse.

If perforation occur in a latent duodenal ulcer, we get the same symptoms as those described when treating of perforation of a gastric ulcer; with this difference, however, that vomiting persists, whilst in gastric ulcers, as a rule, it does not recur after perforation has taken place. Associated pain in the right iliac fossa occurs in cases of perforation of a duodenal ulcer as in those of gastric ulcer, and may lead the physician to

attribute the symptoms to perforation of the appendix. Not long since a patient was admitted into the Manchester Infirmary who, whilst walking in the street, had been seized suddenly with a severe abdominal pain. When I saw him, a few hours after his admission into the hospital, I found him somewhat collapsed, with small quick pulse, and complaining of pain in the right iliac fossa; he had vomited several times since his admission; he told us that he had suffered for some time from indigestion and from pain in the epigastric region. It was clear from the symptoms that he was suffering from a perforation in the intestinal tract. The previous symptoms of indigestion and epigastric pain pointed to the existence of a gastric ulcer, but the persistent vomiting was against this diagnosis; the pain over the right iliac fossa, on the other hand, suggested perforation of the appendix. The patient died some hours after admission, and at the autopsy we found perforation of a duodenal ulcer.

The last group of cases are those in which an ulcer, not having itself given rise to any definite symptoms, leads to narrowness of the duodenum, and causes symptoms similar to those of stenosis of the pylorus; or in which an ulcer, situated over the outlet of the common bile-duct, by its cicatrisation causes persistent jaundice from contraction of the duct. The first class of cases may be indistinguishable from stenosis of the pylorus; in other cases, however, the examination of the gastric juice or of the vomit will show that the obstruction is situated in the duodenum, for the gastric juice will have an alkaline reaction, will contain bile, and will digest fibrine.

Stenosis of the bile-duct due to duodenal ulcer will in most cases be indistinguishable from the symptoms produced by an impacted gall-stone; occasionally, however, a correct diagnosis is possible. A brief account of such a case may be of some interest. In September 1893, a patient was admitted into the Manchester Infirmary suffering from slight jaundice which had lasted for about six months. About two years before his admission he had been troubled with pain after food, and twice had vomited large quantities of blood; he had also passed blood by the bowels, and was told that he had an ulcer in the stomach. Since then he had occasionally suffered from pain, which occurred several hours after meals, but not from vomiting; the jaundice came on gradually, and after it had lasted for some days he had a severe pain which lasted a short time and then subsided; therewith the jaundice also diminished, but did not altogether disappear; and these events recurred from time to time. The patient remained in the hospital for several months, and during this time he had several attacks of pain, with an increase of the jaundice: his general health remained good, and he left the Infirmary to follow his work. I have seen the patient again recently: he is still slightly jaundiced; the pain occurs now only every two or three months, and is much less severe; his general health is so little interfered with that he has not ceased to follow his occupation. In other similar cases (Perry and Shaw) the jaundice became very

intense and the patient much emaciated. The interval from the onset to the fatal termination varied from some months to some years.

The *treatment* of duodenal ulcer and its complications is the same as that of gastric ulcer.

Surgical treatment is indicated when perforation occurs, and may be carried out (75) when a subphrenic abscess arises, occasionally when there is stenosis of the duodenum, and in those cases in which the common bile-duct is narrowed or obliterated. In the latter cases cholecystenterostomy may accomplish a complete cure. This occurred in a patient under the care of my colleague, Mr. Thomas Jones, at the Manchester Infirmary, in whom jaundice had existed for several months; and in whom there was a narrowing of the common bile-duct, due either to ulcer or gall-stone. Other similar cases are recorded by English and Continental observers.

JULIUS DRESCHFELD.

REFERENCES

For the earlier bibliography of duodenal ulcer the reader is referred to BUCQUOY, *Arch. gén. de méd.* 1887.

1. ABERCROMBIE. *Researches on the Diseases of the Stomach and Intestinal Tract*, 1830 (3rd ed. 1837, p. 19).—2. BAILLIE, M. *Morbid Anatomy of the Human Body*, London, 1803 (5th ed. 1818, p. 149).—3. BAMBERGER, H. *Die Krankheiten des chylos-poetischen Systems*, 1864.—4. BARLING. *Brit. Med. Jour.* June 15, 1895.—5. BOAS. *Diagnostik und Therapie der Magenkrankheiten*, 1895.—6. BOETCHER. *Dorpater med. Zeitsch.* 1874.—7. BRINTON, J. *Ulcer of the Stomach*, 1837.—8. BROWN-SEQUARD. *Comptes rend. de la Soc. de Biologie*, 1851, 1861, 1871.—9. CHIARI. *Prag. med. Woch.* 1885, No. 27.—10. CHVOSTEK. *Wien. med. Jahrb.* 1883.—11. COHNHEIM. *Vorles. über all. Path.* vol. ii. p. 53.—11*a*. COMTE. *The Medical Week*, 1895, p. 457.—12. CORNIL. *Manuel de l'histolog. patholog.* 1882.—13. CRUVEILHIER. *Atlas of Pathological Anatomy*, 1838.—14. CURLING. *Medico-Chirurg. Trans.* vol. xxv. p. 260; 1842.—14*a*. DA COSTA. *Medical Diagnosis*, Philadelphia, 1887.—15. DAHLERUP. Abstract in Canstatt's *Jahresb.* 1842.—16. DEBOVE and RENAULT. *Ulcère de l'estomac*, p. 16.—17. *Ibid.* p. 124.—17*a*. DECKER. *Berl. klin. Wochens.* 1887.—18. DEROUET. *Thèse de Paris*, 1879.—19. DIETRICH. See Debove and Renault, p. 138.—20. DONKIN. *Lancet*, 1891.—20*a*. DOYEN, E. *Centralblatt für Chirurgie*, July 6, 1895.—20*b*. *Idem. Traitement chirurgical des affections de l'estomac et du duodénum*, 1895.—21. DRESCHFELD. *Med. Chronicle*, vols. xiii. xiv. xv.—22. DUPLAY. *Arch. gén. de médecine*, 1881.—23. DUSSER. *Thèse de Paris*, 1890.—24. EBSTEIN. *Arch. f. exp. Path.* 1874, p. 183.—25. EWALD. *Klinik der Verdauungskr.* 1890-3. Translated by the Sydenham Society.—26. FENWICK, S. *Journal of Path. and Bact.* vol. i. June 1893.—26*a*. *Idem. Disorders of Digestion in Infancy*, 1897.—26*b*. FINNY. *Trans. Acad. Med. Ireland*, 1886.—27. FISCHER, O. *Dissertation.* Jena, 1893.—28. FLEINER. Abstract in *Brit. Med. Jour.* vol. ii. 1896.—29. FOX, WILSON. *Path. Trans.* vol. xix. p. 239.—30. GALLIARD. *Thèse de Paris*, 1882.—31. GERHARDT, C. *Deutsch. med. Wochenschr.* 1880, No. 18.—32. GOULD. *Brit. Med. Jour.* Oct. 28, 1894.—33. GRASSIUS. Quoted by Debove and Renault, p. 2.—34. GRIFFINI and VASSALI. Ziegler's *Beiträge für path. Anat.* vol. iii. p. 426.—35. GUNSBERG. *Arch. f. phys. Heilkunde*, 1852.—35*a*. HABERSHON. *Diseases of the Abdomen.*—36. HAEBERLIN. *Deutsch. Arch. f. klin. Med.* 1889.—37. HAUSER. *Deutsch. med. Wochenschr.* 1890.—38. HAYEM. Hayem et Winter, *Du chémisme stomach.* 1891.—38*a*. HEAD. *Brain*, Spring number, 1893.—39. HEINEKE. *Handbuch der speciellen Therapie*, edited by Penzoldt and Stintzing, vol. iv. p. 449.—39*a*. HIRSCHFELD. *Deutsch. Zeitsch. f. klin. Med.* vol. xxviii. p. 176.—40. HITZIG. *Berl. klin. Wochenschr.* 1892, No. 49.—41. HUNTER, WM. *Path. Trans.* 1890, p. 105.—41*a*. JACKSCH. *Ueber Acetonurie und Diaceturie*, 1883.—42. JAWORSKI. *Deutsch. med. Wochensch.* 1886 and 1887; and paper in *Zeitsch. f. klin.*

Med.—43. JAWORSKI and KORCZINSKI. *Deutsch. Arch. f. klin. Med.* 1890-91.—44. JOWERS. *Lancet*, March 2, 1895.—45. KLEBS. *Path. Anatomie*, vol. i.—46. KUSTER. *Deutsch. med. Wochenschr.* 1894, No. 24.—47. LANCEREAUX. *Gaz. des hôp.* 1876.—48. *Idem. Traité de Syphilis*, p. 248.—49. LANG. *Wiener med. Presse*, 1885.—50. LANGERHANS. *Virchow's Archiv*, vol. cxxiv.—51. LAW. *Dublin Hosp. Gaz.* vol. ii. p. 51.—52. LETULLE. *Acad. des Sciences et soc. méd. des hôp.* 1888.—52*a*. LORENZ. *Zeitsch. f. klin. Med.* vol. xix. p. 34.—53. MARTIN, SIDNEY. *Diseases of the Stomach*, p. 398.—54. *Ibid.* p. 418.—55. MATTHES. *Verh. d. Congress f. innere Med.* 1893, p. 426.—56. *Idem. Cbl. f. klin. Med.* 1894, No. 1.—56*a*. *Treatise on the Surgery of the Alimentary Tract.*—56*b*. NEUBAUER and VOGEL. *Analyse des Harnes*, 9th edition, 1890, p. 473.—57. NEUMANN. *Syphilis*, Nothnagel's *Cyclopædia of Medicine*, vol. i. p. 360.—58. *Ibid.* vol. i. p. 351.—59. PANUM. *Virchow's Arch.* vol. xxv. p. 491.—60. PAVY, F. W. *Phil. Trans.* 1863.—61. PENZOLDT. *Treatment of the Ulcer*, Penzoldt's *Handbuch d. Therapie*, vol. iv.—62. PERRY and SHAW. *Guy's Hosp. Reports*, 1893, vol. i.—63. PREYER, S. *Sammlung physiolog. Abhandlung.* 1879, ii. Heft 6.—64. QUINCKE. *Deutsch. med. Wochenschr.* 1882, p. 79.—65. RASMUSSEN. *Centralb. für med. Wissen.* 1887.—66. *Report of the Royal Commission on Tuberculosis*, Part ii. Inquiry ii.—67. RIEGEL. *Zeitschrift für klin. Med.* vol. xii. p. 434.—68. RITTER. *Zeitsch. f. klin. Med.* 1888.—69. ROKITANSKY. *Med. Jahrbucher des Oesterreichen Staates*, 1839.—70. ROSANOW. *La semaine médical*, 1890.—70*a*. ROSENFELD. *Deutsch. Med.* 1885.—71. ROSENHEIM. *Deutsch. med. Wochenschr.* 1888, No. 22.—72. ROSS. *Brain*, January 1888.—73. SCHÄFER. *Deutsch. med. Wochenschr.* 1892, No. 46.—74. SCHIFF. *Unters. zur Physiol. d. Nervensyst.* 1855, p. 406.—75. SHIELD, M. *Internat. Med. Mag.* Jan. 1895.—76. SIEBERT. Casper's *Wochensch. f. Heilk.* 1852.—77. SILBERMANN. *Deutsch. med. Wochenschr.* 1886.—78. SOHLERN, v. *Berl. klin. Wochensch.* 1889.—79. STINTZING. *Handb. der spec. Therapie*, edit. by Penzoldt and Stintzing, vol. iv.—80. VAN DER VELDEN. *Deutsch. Arch. f. klin. Med.* vol. xxii. p. 369.—81. VANNI. *Lo sperimentall*, 1889.—82. VIRCHOW. *Virchow's Archiv*, vol. v. p. 362.—83. VULPIAN. *Leçons sur l'app. vasom.* vol. i. p. 216.—84. WAGNER. *Arch. f. Heilkunde*, vol. iv. p. 226.—85. WELCH. Pepper's *System of Medicine*, vol. ii. p. 497.—86. WILKS and MOXON. *Path. Anat.* 1889, p. 404.—87. WILLIAMS. *Lancet*, 1874.—88. ZWEIFEL. *Deutsch. Arch. f. klinisch. Med.* vol. xxxix. p. 349; 1886.

J. D.

TUMOURS OF THE STOMACH

PRIMARY CARCINOMA is so much the most frequent and important of the tumours of the stomach that indeed it is the only form of much clinical interest; it will therefore be considered first. Towards the end of this article, secondary carcinoma, carcinoma infiltrating from some neighbouring organ, sarcoma, fibroma, fibromyoma, adenoma, and non-malignant villous tumours will be briefly described.

The stomach is a very common seat of primary carcinoma; according to some authors no organ in the body is more frequently affected. I find at Guy's Hospital that in the last ten years it caused the death of 60 patients, that is to say, in hospital practice it is responsible for 1·5 per cent of all deaths. Authors are agreed that it is commoner in men than in women; some give the proportion as 3 to 2, others as 2 to 1. The post-mortem records at Guy's Hospital give a proportion of a little over 2 to 1. It is a disease of the later half of life. Dr. Perry and Dr. Shaw found the average age at death to be 52·1 years. Of their cases the youngest was 32 and the oldest 81. According to Brinton,

three-fourths of the cases occur between 40 and 70; between 50 and 60 is the most fatal decade. It has been alleged that sometimes cancer of the stomach is met with in young subjects; but it is very probable that some, at least, of these cases were instances of sarcoma, for among Perry and Shaw's series of fifty cases, all examined histologically, no case of cancer occurred in a patient younger than 32; but of four cases of sarcoma one patient was 15 and another 18 years of age. Dr. Norman Moore, however, has recorded an undoubted case of carcinoma of the stomach in a girl æt. 13; but he says that when he described this case no other had then been recorded at so young an age.

Etiology.—We know little of this subject. The only fact that bears on it is that some cases of gastric carcinoma grow from a simple ulcer. Various authors give the percentage of cases of ulcer of the stomach which are followed by cancer as between 5 and 9. When we bear in mind that ulcer of the stomach is more common in women, at the lesser curvature, and on the posterior surface, while cancer is commoner in men and at the pylorus, it is unlikely that a previous ulcer can be of much importance as a cause of cancer. In so far as there may be any truth in this alleged connection it can only be that damaged structures are more apt to become the seat of cancer (*vide* p. 538). In Guy's Hospital Museum there are two specimens of carcinoma beginning in a gastric ulcer; in both, the growth is a spheroidal-celled carcinoma; both patients were women, one aged 39, the other 65. As the pylorus is the narrowest part of the stomach, it is the part most likely to be irritated by the passage of indigestible masses of food; and this may explain the greater prevalence of cancer at the pylorus. Brinton found the growth at the pylorus in 219 out of 360 cases; and Habershon gives the following table, showing the position of the growth in 79 cases which he examined at Guy's Hospital:—

Pylorus	in 41	Centre	in 4
Lesser Curvature	„ 11	Multiple	„ 1
Cardia	„ 10	Greater Curvature	„ 1
Anterior Wall	„ 5	Cardia and Pylorus	„ 1
General	„ 5	Not stated	„ 1

Perry and Shaw found the pylorus involved in 70 per cent of the cases.

There is no evidence that chronic indigestion leads to cancer of the stomach. Sometimes a hereditary history is well marked. Occasionally we see carcinoma in a patient affected with phthisis, but I do not know that this well-known association is especially marked when the cancer grows in the stomach.

Symptoms.—These begin very gradually and insidiously, and have commonly been present some time before the patient consults a doctor. He usually says that for the last few weeks, or it may be longer, he has been troubled with indigestion, one symptom of which is often pain. So common is this mode of onset that gastric carcinoma should always be suspected in any patient who, after the age of 50, becomes chronically dyspeptic for the first time in his life. Many mistakes are made because

this rule is forgotten. When the patient is cross-questioned he usually admits that lately he has felt weak, has lost flesh, and has become pale. Still, as the total number of cases of gastric carcinoma is large, it is by no means rare to meet with instances in which it has never been suspected till secondary growths are perceptible, usually in the liver; and then again there are the exceptional cases in which the onset is quite sudden, the patient having previously been in excellent health. At the early stage of the disease there may be nothing specially noteworthy about the dyspeptic symptoms; but the loss of appetite and repugnance to food may be well marked, and these are important early signs. The tongue is furred, the patient complains of sense of heaviness in the gastric region, and he often suffers from pain and eructations.

The composition of the gas regurgitated into the mouth is variable. As it is usually derived from swallowed air it contains a large amount of nitrogen, often 45 per cent; the oxygen is often reduced to about 7 per cent, for much of it is probably absorbed in the stomach. There is usually a large amount of carbonic acid gas (between 12 and 26 per cent). This is derived partly from the diffusion of carbonic acid gas from the blood into the stomach, and partly from the fermentation which goes on in the stomach in many forms of indigestion, but particularly in that due to cancer, for the secretion of the cancerous stomach is often deficient in hydrochloric acid, which is powerfully antiseptic, and, owing to the pyloric obstruction, the food is often retained a long time; these two conditions are very favourable to the growth of micro-organisms in the stomach. Excellent accounts of all we know about the formation of gas in the stomach have been recently given by Sidney Martin and by M'Naught. The chief fermentative processes are lactic acid fermentation and butyric acid fermentation. There are many micro-organisms which form lactic acid from carbohydrates, and this by a further fermentation may be transformed into butyric acid, carbonic acid, and hydrogen. There are many organisms also which themselves form butyric acid from carbohydrates. Other much less important processes are alcoholic fermentation with the formation of carbonic acid as a by-product (this at the high temperature of the stomach cannot be very active), and actual putrefaction, which in all probability very rarely takes place in the stomach. In some cases it is probable that gases (such as marsh gas and sulphuretted hydrogen), resulting from the decomposition of proteids in the intestine, may regurgitate into the stomach; and lastly, perhaps, both these gases may be derived from food which was already decomposing when swallowed. The practical outcome of all these processes, which are little understood in all their details, is, as we have already seen, that the gas eructated from the stomach often contains, in addition to nitrogen and oxygen, more carbonic acid than that which diffuses from the blood or is swallowed; and it contains from 20 to 32 per cent of free hydrogen, and sometimes marsh gas (when it may be inflammable) and sulphuretted hydrogen. The following table shows the usual composition of the eructated gas:—

Nitrogen	35–45 per cent
Oxygen	6–10 „ „
Carbonic acid gas . . .	12–26 „ „
Hydrogen	20–32 „ „
Marsh gas (sometimes) . .	3–10 „ „
Sulphuretted hydrogen (sometimes).	

Some of the putrefactive and butyric acid micro-organisms are anaerobic; and it may be that, if there be much fat in the food, the presence of fat floating on the surface of the gastric contents will allow these species to flourish. To show the difficulty of the subject we may quote M'Naught's conclusion that hydrogen, marsh gas, and carbonic acid gas may all form even when there is a considerable amount of hydrochloric acid in the stomach.

Pain of some sort or another is an almost constant sign, and is responsible for the drawn look, expressive of much suffering, which the face of a patient suffering from cancer of the stomach usually shows. It is present at some period of the illness in about 90 per cent of the cases. There are two varieties of it, namely, that due to the indigestion, and that due to the cancerous growth; but it is very difficult to separate them. When, however, the pain is principally between the shoulders, and is benefited by taking food, it is probably dyspeptic. The pain characteristic of the cancer itself may be very severe; it is principally felt in the epigastric region, but it may radiate widely. Except in the early stages it is continuous, and may not be altered by the ingestion of food; but, independently of this, it often varies in severity from time to time, and may even be paroxysmal. Patients describe it differently, but generally it is either a wearing pain, or it shoots; in the latter case it is agonising. It is usually more severe than the pain of an ulcer, and it is almost always increased by pressure.

The stomach probably gets its sensory nerve-supply from the 6th, 7th, 8th, and 9th dorsal segments; the 6th and 7th furnishing the cardia, and the 9th the pylorus. Hence, referred pain from a gastric carcinoma is often felt over those areas of skin which, as Dr. Henry Head has shown in his well-known paper, correspond to these dorsal segments; that is to say, in front from just below the nipple to the umbilicus, and behind from just below the 5th to just below the 12th dorsal spine. Further, the cutaneous tenderness—generally best demonstrated by pressure with the head of a pin, which, if the tenderness be excessive, makes the patient flinch or even cry out—may often be elicited over these areas when the stomach is diseased. Each area has points which are more sensitive to this pressure than the rest of the area. Those for the 6th dorsal area are just under the nipple and at the angle of the scapula; those for the 7th are near the tip of the ensiform cartilage and below the angle of the scapula; those in front, for the 8th and 9th areas, are both in the nipple line; the 9th is on the costal margin, the 8th above it; behind the 8th the tender area is below the 7th, and the 9th below the 8th. When the stomach is

diseased these tender spots are very evident; but cutaneous tenderness is of less value in gastric carcinoma than in other diseases of the stomach, for cases of malignant disease are complicated by the presence of tender areas due to secondary deposits in other organs; moreover, in long-standing and exhausting diseases, pains often radiate far beyond the areas usually associated with the affected organ. Many patients complain of pains in the head, and the scalp should be tested for local tenderness. Head has shown that there are painful and tender areas on the scalp which correspond to dorsal cutaneous areas; and they may be the seat of pain and tenderness when either is felt in its corresponding dorsal area. The occipital area corresponds with the 10th dorsal, the parietal with the 9th. The area, which is triangular with its base at the middle line of the head and its apex in front of and above the ear, corresponds to the 8th dorsal; and the temporal area just behind the outer part of the eyebrow corresponds to the 7th.

When these dyspeptic symptoms have lasted some time, the patient begins, in most cases, to complain of nausea, commonly succeeded by vomiting, which, unlike that of most other gastric affections, does not relieve the pain. This symptom is present in 87 per cent of the cases. It is most troublesome when there is some stenosis of the pylorus; and it bears more relation to the volume of the contents of the stomach than to the ingestion of food. The vomited material is faintly acid, dark brown, and often smells very disagreeably when it has undergone butyric acid fermentation or, in rare cases, putrefaction. Under the microscope pieces of undigested food (which may have been swallowed days before), crowds of micro-organisms—especially sarcinæ, blood-discs fresh and altered, and, in excessively rare cases, cells derived from the growth may be seen.

Much attention has lately been directed to the kind of acids present in the gastric contents in cases of gastric carcinoma. The vomit itself may be examined, but it is much better to wash out the contents of the stomach with pure water in the morning after the patient has been fasting during the night, and then to give a little simple food of such a nature as not to cause vomiting. If possible, it should contain a fair amount of proteid; thus egg and milk is often suitable food. An hour or two afterwards the contents may be syphoned out and examined for hydrochloric and lactic acids. Many tests have been recommended for the detection of free hydrochloric acid in the stomach contents. I have always found Günzburg's phloroglucin and vanillin test answer well (*vide* p. 289). Boas' resorcin test is also useful (*vide* p. 290). Tropæolin (00) and congo red may either of them be used as colour tests. Full details of the mode of application are given by Sidney Martin. Toepfer has recently introduced what he alleges to be an excellent test for free hydrochloric acid, and Friedenwald highly approves it. To 10 c.c. of gastric filtrate three or four drops of a 0·5 per cent alcoholic solution of dimethylamidoazobenzene are added; if free hydrochloric acid be present the mixture becomes red. If a quantitative estimate

be desired the mixture is triturated with a decinormal solution of sodium hydrate till it turns yellow; the number of cubic centimetres of the sodium hydrate solution employed for 100 c.c. of gastric filtrate multiplied by 0·00365 gives the percentage of free hydrochloric acid present.

To detect lactic acid Ueffelmann's test may be used (*vide* p. 290). For the further details on the testing of the gastric contents Sidney Martin's work should be consulted.

Although occasionally in the earlier period of the disease cases of cancer of the stomach may be found in which hydrochloric acid may be detected in the gastric contents, yet when the patient first comes under observation it is nearly always absent; I have only once known it present, and it is so very much more often absent than in any other disease that it is important always to search for it. No satisfactory reason for its frequent absence has been given. Free lactic acid is not common in the gastric contents unless the patient be suffering from cancer of the stomach, and in this disease it is often present. Manges found it in 20 out of 21 cases; Ewald found it in 22 out of 24; but Straus found it in 5 only out of 12 non-malignant cases of gastric disease. It is merely the result of the retention and consequent bacterial decomposition of the gastric contents. In doubtful cases it is of the greatest importance to search for hydrochloric and lactic acids.

All the symptoms hitherto described are usually early. When the patient comes under treatment the indigestion and vomiting may lessen, and careful diet and rest in bed may even make him gain a little in weight. Hence he is often buoyed up by false hopes, and the physician may begin to wonder whether his diagnosis is wrong; but it is a good general rule to be very slow to alter the diagnosis of cancer when it has been reached after a careful survey of the whole case. Any temporary improvement passes away, and the symptoms again begin slowly to increase in severity; the vomiting becomes more frequent, and the wasting is more marked.

In about 35 to 40 per cent of the cases there is at some time or another blood in the vomit. This is due to the ulceration of the growth. If the bleeding be slight, the blood may remain in the stomach long enough to be partially digested; the vomit then looks like coffee grounds. On the other hand, the hæmorrhage may be profuse, and a quantity of bright red blood may be ejected at once; but this is not so common as the "coffee-grounds vomit." Melæna is not very frequent, for it often happens that the pyloric obstruction prevents the blood from reaching the bowel. In the extremely rare cases in which the growth is at the cardiac orifice there may be dysphagia.

There is very little to say about the general signs. If the patient live long enough he has the thin, dry wrinkled skin which is so characteristic of cancer; he wastes to a skeleton, and his face is pinched and expressive of great suffering. The loss of flesh in cases of cancer of the

stomach is often more rapid than in that due to malignant disease of other organs; for to the wasting due to the disease itself is added that due to the lack of food, the vomiting, and the imperfect digestion. The patient is pale and sallow, his red blood corpuscles diminish in number, and contain less hæmoglobin than normal; the leucocytes are slightly increased in number as in other wasting diseases. His temperature is low, he is excessively weak, the myotatic irritability is increased, there may be œdema of the ankles, the urine sometimes contains a considerable amount of ethereal sulphates, especially indican, peptonuria may appear, and, towards the end, a little albuminuria.

Whether the growth produce any signs due to mechanical interference depends entirely on its position. As it is usually seated at the pylorus, the pyloric orifice is often narrowed, and consequently the stomach dilates and becomes over-full. An account of the symptoms of dilatation of the stomach is given in another article (see p. 497). A growth at the pylorus commonly leads to constipation, but sometimes in carcinoma of the stomach there is diarrhœa, probably due to the passage of some of the decomposing contents of the stomach through the pylorus; if so, it indicates that the obstruction is not great. The rectus muscle over the growth is often rigid; in fact, constant rigidity of the right rectus, if other symptoms point in the same direction, supports the diagnosis of carcinoma of the stomach. It is frequently possible to feel a malignant tumour of the stomach, but the possibility of doing this clearly depends upon its position, its size, and the thickness and degree of relaxation of the abdominal wall.

To feel a tumour of the stomach to the best advantage the patient should be on his back, with his knees drawn up; he should breathe deeply and keep his mouth open. If now the abdominal muscles are not relaxed his attention may be distracted by making him talk. If it is still impossible to form a satisfactory opinion, it may be necessary to give him some chloroform. The examiner's hand should always be warm, and should be moved slowly and evenly over the abdomen. A malignant tumour of the stomach is hard, tender, and often irregular. It varies very much in size, but it slowly increases. Usually a dull note is obtained over it, but sometimes on deep percussion an obscurely resonant note may be heard. Inflation of the organ with gas may render the tumour more easy to detect. The pylorus can hardly ever be felt in health, so that if this part can be made out by palpation it is pretty certainly diseased. If enlarged it is usually felt under the outer part of the right rectus, or at the right border of it; but although tumours of the pylorus are, as a rule, much more fixed than those in other parts of the stomach, owing to the close attachment of this structure to the liver, yet not infrequently these attachments stretch, and the tumour becomes freely movable over a range of as much, may be, as three or four inches, so as to suggest the presence of a floating kidney; but it is more usual for tumours of the stomach to become more and more fixed, as in the course of time they form more numerous and denser adhesions. A tumour

of the pylorus moves up and down with the liver in respiration, and it is usually impossible, either by palpation or percussion, to separate it from the liver. It must not be forgotten that even an enlarged pylorus is frequently so covered by the liver that it is not to be detected during life. If the growth is in the anterior surface and along the greater curvature, it is more easy to detect, is more mobile under the hand, and alters its position when the stomach is distended with gas; but it does not move so freely with respiration. If, as is sometimes the case, it forms a mass along the greater curvature, we feel a hard, irregular, tender tumour running transversely across the abdomen above the umbilicus. This is very liable to be confounded with a thickened puckered omentum—such as we get in chronic peritonitis of any form, or a growth in the colon, or the thickened lower edge of a diseased liver. Tumours limited to the cardia can seldom be felt.

A tumour of the stomach is rarely large enough to form a visible projection of the abdominal wall; but towards the end of the case, when the patient is very wasted, it may sometimes be seen. The dilated stomach is often visible. If the mass lie over the aorta it may be lifted up and down by the pulsations of this vessel.

Complications.—Secondary deposits in other organs are very common. On an analysis of thirty-eight cases of malignant disease of the stomach, collected by Perry and Shaw, I find that at death no secondary deposits were found in seven cases (18 per cent). They occurred in one organ in fifteen cases; in two organs in eight cases; in three organs in six cases; in four organs in one case, and in eight organs in one case.

All authors are agreed that the lymphatic glands are most frequently affected, deposits being found in them in a little over a third of all cases; the liver is affected in about a third, the peritoneum in about a fifth, the lungs in about an eighth, and the pleura in about a tenth; but deposits may be found anywhere in the body. These secondary deposits frequently cause important symptoms; thus the affection of the liver, or of the enlarged glands in the portal fissure, often leads to fatal jaundice, or the implication of the peritoneum, helped by pressure of some mass on the portal vein, leads to ascites. Secondary growths too often lead to death from bronchitis and pneumonia, and when the pleura is affected there may be pleural effusion.

In cancer of the stomach, as in all wasting diseases, thrombosis is liable to occur. It is most frequently seen in the left saphena, or in the left femoral vein. The wasting may induce a fatty liver and a fatty heart, and death may be due to cardiac failure; or the patient may become so thin that, if the nursing be inefficient, a bed-sore may form and hasten his end.

The growth in the stomach by its ulceration may lay open a large artery; death may then result with profuse arterial hæmatemesis, but this is a rare termination. We have in Guy's Hospital Museum a specimen in which the splenic artery was thus laid open. Abscesses not due to actual perforation may arise in connection with the growth. Often

these are only found at the necropsy; but there may be a collection of pus in the subphrenic region large enough to be detected during life. Sometimes cases are seen in which rigors occur. These are usually due to suppuration; but it is said that both rigors and pyrexia may occur without it. Not infrequently the patient appears too weak to show any signs of suppurative processes. The drowsiness and the delirium which have been observed may perhaps be due to toxic absorption from the foul contents of the stomach; indeed, we do not know how far such a cause may be concerned in the production of general symptoms.

Perforation occurs in about 4 per cent of the cases; usually it takes place into the peritoneum, and if so is fatal. If the patient be very weak and exhausted at the time of perforation he may show no signs of the consequent peritonitis. If perforation take place into adhesions formed around the growth, a local abscess forms without general peritonitis; this may lead to a communication between the stomach and colon. Thus fæcal vomiting may be set up with a horrible taste in the mouth and odour of the breath; but if the stomach be constantly distended and full of fluid, there may be no passage of the contents of the colon into it. In about a third of the cases of gastro-colic fistula undigested food may be observed in the fæces; whenever food appears soon in the fæces in an undigested state the condition is termed lientery, but this may occur when there is no fistulous communication. Other perforations are extremely rare; but there may be a communication between the stomach and duodenum, or a gastro-cutaneous fistula may form (*vide* p. 572).

Termination.—It is clear that several of these complications may kill the patient; but not uncommonly he appears to die slowly from sheer weakness, lying during the last few days of his life quite still, almost pulseless, and with hardly any respiratory movements. I have often noticed that, as Fagge points out, many of the symptoms diminish towards the end; the pain is less, and the vomiting stops; but I do not know that this remission is peculiar to cancer. Near the end of any exhausting disease all the functions of the body, even the conducting power of the nerves, are depressed; consequently no pain is felt, and reflex acts fail. I have seen cases of perforation in typhoid fever and in cancer of the cæcum in which during life there had been no signs to lead me to suspect what had happened.

Pathology.—The naked-eye appearances of malignant disease of the stomach vary greatly, and depend chiefly on the extent of the growth —which is sharply limited by the pylorus—the amount of fibrous tissue in it, and the degree of contraction or ulceration. There may be so much fibrous tissue that the new growth appears exactly like a dense, hard, innocent fibrous tumour, and its true nature is only revealed by an examination of the lymphatic glands. Such tumours are often sharply defined, are frequently limited to the pylorus, show much hypertrophy of the muscular coats, and yield no juice on scraping. There are all degrees between these and those rapidly-growing ulcerated masses which, sloughing, sup-

purating, or bleeding, destroy all the coats of the stomach beyond recognition, and form such horrible-looking specimens. A common intermediate form is a hard, well-defined tumour usually invading the pylorus, having a distinct edge, and extending along the lesser curvature. On section the whitish new growth in the submucous coat stands out in very marked contrast to the darker hypertrophied muscular layer, especially at the outer part of the tumour; for at the centre the muscle is often destroyed by the carcinomatous tissue, or has several whitish strands of growth running parallel to its muscular fibres. The peritoneal coat is opaque and puckered, and the total thickness of the stomach wall may be half an inch or an inch. If the growth has undergone colloid degeneration, or be a villous tumour, its appearance will be modified accordingly. When the whole of the wall of the stomach is affected, it forms what is known as the india-rubber bottle stomach, in which case the walls are usually much thickened—they may be half an inch thick—and the new tissue is so contracted that the stomach is very small (*vide* p. 441). In a case that occurred at Guy's Hospital it was seven inches long, and its cavity was so contracted that it only measured two inches across at its widest part. The growth may adhere to and invade the liver, the pancreas, the spine, the intestine, or the abdominal wall; but adhesions to the liver, intestine, and pancreas are much the commonest, and these organs are often extensively invaded. To complete the picture we must imagine that in most cases there are secondary deposits, that fistulous communications sometimes form, and that ordinarily when the growth is at the pylorus the stomach is much hypertrophied and dilated.

Histology.—Drs. Perry and Shaw have so recently written a very full article, giving an account of the material at my disposal, namely, that in the Museum at Guy's Hospital, that I cannot do better than base my description largely on their paper. They analysed fifty cases of malignant disease of the stomach, and they divide the carcinomas into cylindrical carcinoma and spheroidal carcinoma. The latter is three times as common as the former. The tumours, called by some authors destructive adenoma, are included among the cylindrical carcinomas, for in most specimens it is easy to see many transitions between the two forms. Of eleven cylindrical carcinomas five were limited to the pylorus and encircled it; two involved the lesser curvature and cardia; one affected the entire organ; two implicated the pylorus and a good deal of the stomach besides, and one was limited to the posterior wall. Thirty cases of spheroidal carcinoma were described, and the different regions of the stomach were for the most part affected in the same proportion as in cyclindrical carcinoma, the pylorus being involved in about 70 per cent of both classes. The only possible exceptions concerned growths limited to the cardia, of which there were three, and all were spheroidal carcinoma; and growths which involved the entire organ, seven out of eight of which were spheroidal carcinoma. Age and sex, apparently, had no influence on the form of carcinoma; and no difference could be made out between the two forms as regards the probability of secondary deposits or the organs in

which they would be deposited; the figures already given (p. 561) will apply, therefore, to either form.

Colloid degeneration appears to be much more common in spheroidal than in cylindrical carcinoma; for it was observed in one out of twelve cases of the cylindrical, and in nine out of thirty-two cases of the spheroidal kind. Two of the whole series of fifty cases were villous malignant growths, both cylindrical carcinoma; circumscribed globular or subglobular tumours also usually belong to this variety, but diffuse infiltrating growths, especially when accompanied by contraction, are almost certainly spheroidal carcinoma. Either variety may be medullary or scirrhous, both lie mostly in the submucous coat, but the medullary variety destroys the coats and projects externally more than the scirrhous; and on the whole, medullary cancer is more common at the cardiac than at the pyloric end. Eight (16 per cent) of the cases fall under the heading of india-rubber bottle stomach, the wall being universally indurated and the cavity of the viscus mostly contracted. In these cases the mucous membrane is nearly always smooth, the submucosa is thickened, especially towards the pylorus, the muscular coat is hypertrophied, and the serous coat opaque. Seven of these eight cases of india-rubber bottle stomach were cases of spheroidal carcinoma, with much fibrous tissue, the amount varying with the degree of contraction of the organ. There may be so much of it that many slides must be examined before any cancer cells are found; but even in cases in which they are so few that no section happens to reveal them, the carcinomatous nature of the tumour is betrayed by an examination of the lymphatic glands. Perry and Shaw believe that all universal fibrous induration of the stomach not due to swallowing corrosives is caused by scirrhous carcinoma. And, further, a considerable number at any rate of cases of so-called non-malignant scirrhus of the pylorus (fibrous pylorus) not due to corrosive poisoning or the contraction of an ulcer, are really scirrhous cancer. Certainly I am disposed to agree strongly with both these propositions. The only exception I know is that in the chronic inflammatory thickening which occurs around the gall-bladder in some cases of gall-stones the pylorus may be very thick and fibrous (compare, however, pp. 441 and 497).

Perry and Shaw paid particular attention to the origin of carcinoma in those cases in which it occurs at the cardiac end of the stomach only. As I have already said, they found four specimens; and they show that these were all spheroidal carcinoma. But among over twenty cases of œsophageal carcinoma examined, not one was spheroidal; it is highly probable, therefore, that these growths began in the stomach and spread into the œsophagus rather than, as Fagge supposed, that they spread the other way (*vide* p. 373).

Diagnosis.—It may be difficult to distinguish between ulcer and carcinoma of the stomach; but the subjects of ulcer are more often women, and are younger than sufferers from carcinoma. They commonly give a history of previous similar attacks; or at any rate they say they have before suffered from indigestion; but malignant disease of the stomach

often occurs in persons who have never had indigestion. The pain of ulcer is made worse by food, and is relieved by vomiting; in cancer this is less common. Wasting is a less marked symptom in ulcer, and the patients do not so often complain of loss of appetite and repugnance to food. The absence of hydrochloric acid and the presence of lactic acid and sarcinæ are strongly in favour of cancer; and it is rare for an ulcer, by its contraction, to narrow the pylorus and so produce all the symptoms of dilatation of the stomach. The detection of a tumour which can be definitely located in the stomach, or the presence of secondary growths, is of course conclusive. Osler quotes the case of a man in whom a small nodule in the anterior abdominal wall was associated with gastric symptoms of uncertain origin. It was excised, found to be carcinoma, and a diagnosis of gastric carcinoma was founded upon it which proved to be correct. Profuse hæmorrhage and bright redness of the blood are in favour of ulcer, and the patients affected with this malady rarely have the appearance of those suffering from cancer. Lastly, a duration of more than eighteen months is much in favour of ulcer.

In the early stages the diagnosis between cancer and chronic catarrhal gastritis is often impossible; but, as already remarked, when symptoms of indigestion come on for the first time after the age of fifty they are nearly always due to cancer; but under thirty hardly ever. After what has been said about carcinoma, the reader will easily be able to perceive the signs which, in a later stage of cancerous disease, will prevent a mistake.

From a practical point of view the most difficult question is whether a patient who is obviously ill and anæmic is suffering from phthisis, pernicious anæmia, or from a latent carcinoma of the stomach.

I do not think any useful purpose will be served by going over the points of diagnosis, for each case must be judged on its own evidence. The best way to avoid a mistake is to remember the possibility of it. Great care may be necessary to determine whether a tumour in the abdomen springs from the stomach or not. If it is in the pyloric region, it may be attributed to the liver or gall-bladder; or it may be that from stretching of the attachment of the stomach it is mistaken for a movable kidney. The pylorus may in such cases be felt as a tumour below the umbilicus, and may be moved three or four inches with the hand. An excellent case in point is figured by Osler. Great attention must be paid to all the symptoms of the individual case. If the stomach be dilated, or gas can be felt on pressure to bubble through the tumour, it is in the pylorus. Growths in the body of the stomach often form a hard solid mass running transversely across the abdomen, so that they are very difficult to tell from the edge of the liver, a thickened puckered omentum, a tumour of the colon, or a fæcal accumulation. In such cases it is of the greatest possible importance to observe the precise position of the tumour, its extent, and its relation to the edge of the liver; and to distinguish the gastric resonance from that due to the colon. Inflation of the stomach will help us much; and it is often wise not to give too positive

an opinion until the bowels have been well opened by an enema. In rare cases it has been a matter of difficulty to distinguish between an enlarged spleen and a tumour at the cardiac end of the stomach.

Prognosis.—A malignant tumour of the stomach must sooner or later kill; and life is rarely prolonged more than eighteen months, or at the outside two years, after the patient is first led to consult a doctor for the symptoms of this disease. The duration of the greater number of cases is much less than eighteen months, and often is less than a year. Much depends upon the rate of growth of the tumour and the rapidity with which secondary growths form.

Treatment.—This can only be palliative. In most cases subcutaneous injections of morphine are necessary sooner or later, and they should be given whenever pain is severe. It is cruel to withhold them from a man who is suffering agony and has only a few months to live. When the dilatation of the stomach is well marked much relief is afforded by frequent lavage, and by not allowing much fluid to be drunk. In the early stage, when the loss of appetite is a prominent symptom, benefit may follow the use of an acid bitter mixture. For example, ten minims of each of dilute nitro hydrochloric acid and tincture of nux vomica, with some compound tincture of gentian, or spiritus armoraciæ compositus, given before meals, will help the appetite and compensate the deficient secretion of acid. A glass of sherry and bitters before a meal is of use in the same way. As a rule, it is difficult to prevent fermentation by drugs; but carbolic acid, naphthol, mineral acids, and salicylic acid may be tried, although most of these, if given in sufficient doses to prevent fermentation, irritate the stomach; on the whole, perhaps, salicylic acid is the best of these. Careful attention must be paid to the diet. The food should be given in small quantities at frequent intervals, and generally the feeding resolves itself into giving what the patient finds he can best digest. While on the one hand the food should be simple and capable of easy digestion, such as peptonised milk, on the other hand it should not be such as will easily undergo fermentation; small quantities of meat and jelly are therefore useful, but in different cases the physician has to deal with such varying degrees of failure to digest and liability to decomposition that no detailed rules can be laid down.

Surgical interference in cases of cancer of the stomach is not usually considered unless the growth prevent the passage of the gastric contents through the pylorus; and even then it is rarely justifiable, for experience has shown that by the time this stage has been reached there are many secondary deposits, and the patient is hardly ever able to stand a severe operation. The operations that have been performed with the object of overcoming pyloric obstruction are as follows:—

The simplest method is to open the abdomen, stitch the stomach to the abdominal wall, and a few days later to open the organ by an incision parallel to its long axis, and through this opening to scrape away parts of the cancer with a curette or some suitable instrument. This method is not to be recommended; the relief is very transient and the bleeding may

be dangerous. Some surgeons have simply excised the pylorus; this is a very difficult operation, for it is not easy to drag the duodenum and the stomach together, and even if this be achieved the operation is very severe. Out of sixty-six cases quoted by Treves, fifty died soon after the operation, and in all the remainder there was speedy recurrence. We are not surprised, therefore, that the operation is now very rarely performed; probably it is never justifiable. In a few cases the duodenum or jejunum has been brought to the surface of an abdominal wound, and stitched there, and when adhesion has taken place the bowel has been opened and the patient fed through the opening; but this operation too has been discarded, as the patients have soon died after it. A much better operation is to open the abdomen and form a fistulous opening between the stomach and some prominent coil of the jejunum. But this operation—gastro-jejunostomy—is usually also quickly followed by death; and even if the patient survive some time, the opening will probably slowly contract. Still, if it should be decided that the patient is suffering mainly from the pyloric obstruction, and that he is in such a condition that an operation is justifiable, then gastro-jejunostomy should be performed; by its means considerable suffering may be avoided, and the life of the patient probably prolonged for a few weeks or even months. Quite lately a carcinomatous pylorus has been excised, the ends of the duodenum and stomach closed up, and then, at the same operation, a gastro-jejunostomy performed; it is possible that future experience will show that this is good treatment for cases in which at the time of operation few secondary growths can be felt, and perhaps it will come to be the accepted treatment early in the case before pyloric obstruction has become established. Loreta's operation and pyloroplasty are not justifiable except for non-malignant stricture of the pylorus.

SARCOMA OF THE STOMACH.—I have incidentally mentioned that Drs. Perry and Shaw, out of fifty cases of primary malignant disease of the stomach, found that the growth was sarcomatous in four. In all it was of the round-celled variety. The new growth was most extensive in the submucous coat, but the mucous, serous and muscular layers were affected. The mucous was only slightly ulcerated, but the muscular was extensively destroyed. In both these points it will be noticed that a sarcoma differs from a carcinoma of the stomach. In all the tumour was sharply defined, and in all the pyloric end was affected. In two the growth was continued into the duodenum. Two were males and two were females. The ages were 15, 18, 38 and 67 years. As the youngest patient with carcinoma was 32, it is probably justifiable to diagnose sarcoma when we have all the symptoms of malignant disease of the stomach in a patient under 30 years of age. In three of the cases secondary deposits were found in other organs.

TUBERCLE OF THE STOMACH.—This is excessively rare, but a boy aged 7 years, thus affected, died under my care. He had tubercular

peritonitis. On the posterior wall of the stomach, rather nearer the pyloric than the cardiac orifice, was an irregular patch of superficial ulceration with sinuous edges and a ragged base. The part of the serous coat corresponding to the ulcer was covered with miliary tubercles, and the lymphatic glands on the lesser curvature were enlarged and caseous. In the Guy's Hospital Museum are two other specimens of tubercular ulcers of the stomach, both from children; a specimen from a man aged 48, who died of tubercular peritonitis, showing a submucous caseous deposit without ulceration; and a specimen from a youth aged 18, who also died of tubercular peritonitis, which shows a small submucous abscess probably tuberculous.

FIBROMA OF THE STOMACH.—The older writers described a fibroma of the pylorus; but probably their cases were either scirrhus with much fibrous stroma, or the pylorus was thickened either from within as a result of a cicatrising ulcer, or from without as a result of inflammation around the gall-bladder and due to gall-stones. I have seen an instance of this last mistake. We have in our Museum a specimen (exhibited by Dr. Pitt at the Pathological Society) taken from a child aged five weeks, in whom all the viscera were normal except that the pyloric opening was so narrowed from considerable hypertrophy of the muscular coat that it would hardly admit a small probe. Finkelstein has recently collected several cases of this rare congenital malady. The children usually die within a few months of birth from vomiting, constipation and symptoms of intestinal obstruction.

FIBROMYOMA OF THE STOMACH. — In very rare instances these innocent tumours, which may be of any size up to a pigeon's egg, are found projecting into the stomach. They consist of unstriped muscle and fibrous tissue; the mucous membrane over them is intact, and so far as we know they are harmless. Sometimes they are pedunculated, when they form one variety of polypus. Two or three may be present in the same patient.

ADENOMA AND PAPILLOMA OF THE STOMACH.—Occasionally small white translucent non-malignant tumours, composed of tubular structures like normal glands, are found in the stomach. Papillomas are either wart-like or polypoid overgrowths of the mucous membrane; they are very closely related to adenoma. Small lipomas may occasionally be seen in the submucous coat.

VILLOUS TUMOURS OF STOMACH.—These are usually malignant; but they may be innocent, and consist of mucous membrane supported on delicate bands of fibrous tissue.

LYMPHADENOMA OF STOMACH. — This is very rare. Dr. Newton Pitt has collected several cases. In the intestine the lymphoid growth

may occur either in the submucous coat, in which case large flat tumours, the mucous membrane of which is not ulcerated, project into the lumen of the bowel; or it may take place in the muscular coat, when the mucous membrane ulcerates and the calibre of the bowel is diminished. Probably the first variety only is met with in the stomach. An increase of lymphoid tissue is always found in other parts of the body.

SECONDARY GROWTHS IN THE STOMACH.—These are rare. Judging by our experience at Guy's, and by other recorded cases, probably about 6 or 7 per cent of malignant growths in the stomach are secondary. The primary growth may be anywhere, and the breast is of course a common seat. I have known an epithelioma of the stomach to be secondary to epithelioma of the œsophagus, and there is in Guy's Hospital Museum a specimen of secondary melanotic sarcoma of the stomach. The symptoms do not differ from those of primary disease of the organ; but often those of the primary growth overshadow them, and the patient dies before the gastric symptoms are very manifest.

FOREIGN BODIES.—Occasionally lunatics and others swallow hair, fibre, or other foreign substances, which slowly aggregate into a mass that may easily be mistaken for a tumour of the stomach.

W. HALE WHITE.

REFERENCES

1. FRIEDENWALD. "Observations on a new test for hydrochloric acid in the gastric juice," *Medical Record*, April 6, 1895.—2. HABERSHON. *Diseases of Abdomen*, 3rd ed. 1879.—3. HEAD. *Brain*, Pts. 61, 62 and 67.—4. M'NAUGHT. "The Gases of the Stomach," *Med. Chron.* Nov. 1893.—5. MANGES. "The Early Diagnosis of Cancer of the Stomach," *Medical Record*, April 27, 1895.—6. MARTIN, SIDNEY. *Diseases of the Stomach*, 1895.—7. PITT, N. "Lymphadenoma of Stomach and Intestines," *Path. Trans.* vol. xl. — 8. OSLER. *Principles and Practice of Medicine*, 2nd ed. 1895. — 9. PERRY and SHAW. "Malignant Disease of the Stomach," *Guy's Hospital Reports*, vol. xlviii.—10. TOEPFER. *Zeitsch. f. physiolog. Chemie*, Bd. xix. Hf. 1, 1894.—11. PITT. "Hypertrophy of the pylorus in an Infant," *Path. Trans.* vol. xliii. p. 63.—12. FINKELSTEIN. *Jahrbuch f. Kinderheil.* Bd. xliii. S. 105 (abst. in *Brit. Med. Journ.* Feb. 6, 1897, p. 21).

W. H. W.

SUBPHRENIC ABSCESS

Definition.—By subphrenic abscess is understood a collection of pus in the hollow of the diaphragm, generally circumscribed in the peritoneal cavity to one or the other side of the falciform ligament of the liver. Retroperitoneal suppuration sometimes usurps a subphrenic position.

The affection arises for the most part in connection with a perforative lesion of some organ below the diaphragm, though not necessarily near it; and, apart from antecedent disease, it has all the general gravity of extensive and possibly fœtid abdominal suppuration. The special importance of a subphrenic, as distinguished from other forms of abdominal abscess, consists in the difficulty of ascertaining its position and relations to the several viscera, and the liability to thoracic complications, which sooner or later too often prove fatal.

Etiology and Symptoms.—The diaphragm is always pushed upwards to encroach upon the cavity of the chest; and the local symptoms are mainly those of embarrassment of the lung.

The physical signs of pus under the diaphragm are very similar to those of a limited pleural effusion or empyema; and when, as frequently happens, the collection is gaseo-purulent, it simulates pyopneumothorax in many ways: in the absence of thoracic complications, however, the distinction is easily made, provided the true state of affairs be suspected. In the former case the collection generally presents itself at a lower thoracic level than would be possible for the degree of pleural exudation observed; in the latter, it often occupies a lower position under the ribs than one to which the diaphragm can possibly descend. Above, the arch of the diaphragm may be directly recognised in the convex upper contour of the collection; while the heart suffers but slight displacement, and this upwards, not laterally.

The respiratory movements of the diaphragm, though suppressed during the first acuteness of the inflammation, are often restored at a later period. When one wing is much raised, as by a collection of gas beneath, it is enabled to contract downwards at a mechanical advantage; whereas in pneumothorax it is extroverted and motionless. Hence there may be a marked inspiratory descent of the lung above, and, in a right-

sided case, of the liver below; and these are conclusive signs of the collection being subphrenic.

When a subphrenic abscess is opened by puncture or incision, the diaphragm being active, the outflow is accelerated during inspiration; the converse of this occurs with fluid in the pleura.

The so-called "diaphragm phenomenon"—a shallow depression which may be seen to cross the corresponding intercostal spaces as the diaphragm contracts in deep inspiration—when present, is a ready indication of its level. This sign, however, is less generally available than those which have previously been mentioned.

The nature of the case is often obscured by thoracic complications. Through the lymphatics of the central tendon of the diaphragm irritative material is readily conveyed upwards from the peritoneal cavity; and pleurisy—dry, serous, or purulent—seldom fails to be an early result. Basic pneumonia may follow. Pericarditis is less common, and is more often due to extension from the pleura than to direct transmission through the diaphragm. In course of time the diaphragm becomes ulcerated or infiltrated by pus; and perforation is the ordinary course of things, the contents of the abscess either breaking into the pleural cavity, or, if adhesions have been formed, finding their way more gradually into the lung.

When the diaphragm is perforated exact diagnosis may be impossible; but a serous or purulent pleurisy—the more ordinary complication—is sometimes indeed an assistance. Thus the recognition of a gaseous below a fluid collection, or the discovery, by puncture of the chest at different levels, that pus is surmounted by serum, or fœtid pus by sweet, is clear evidence that between the two collections is a perfect septum; and this is more likely to be the diaphragm than a false membrane in the pleural cavity.

By far the most frequent cause of subphrenic abscess is perforation of some part of the alimentary canal; in the great majority of cases it is a simple ulcer of the stomach. The abscess thus arising in a free perforation is characterised by the presence of gas, a condition which is known as subphrenic pyopneumothorax; this on account of its frequency and importance will be described here in detail. It is to be observed that a simple abscess may, by secondary communication with the alimentary canal or even with the lung, acquire to some extent the same characters.

When the stomach is perforated by an ulcer the escape of its contents is sometimes so controlled by pre-existent adhesions that the consequences are a slight and gradual leakage and a circumscribed abscess. Such an abscess, however, is not necessarily subphrenic; it may be behind the stomach; between the stomach and colon; beneath the left lobe of the liver, or elsewhere. More often there is a sudden effusion of gas and other matters from the anterior surface or lesser curvature of the stomach immediately under the diaphragm, matters which are limited to this region till encysted by fresh adhesions. Some such limitation, temporary or permanent, is far from uncommon; and there are all degrees of transition

between a peritonitis which, independently of previous adhesions, results in a strictly circumscribed subphrenic abscess, and one which is diffuse from an early stage—a point of some practical importance.

The boundaries of the abscess vary, of course, in different cases; but upon the whole they are remarkably uniform. The ordinary form of abscess is above and in front of the stomach, occupying the left wing of the diaphragm from the falciform ligament on the right to the false ribs on the left. The closure below is effected by adhesion of the stomach and left lobe of the liver to the abdominal wall; or, if the downward extent be unusually great, by similar adhesion of the omentum or transverse colon. The greatest depth is backwards and to the left, whither the pus gravitates and sometimes erodes the spleen.

Occasionally, when the perforation is near the pylorus, the abscess is to the right of the falciform ligament, between the right lobe of the liver and the diaphragm. The ligament is sometimes broken through by the suppuration, or an independent abscess may form on each side of it. All such cases, however, are exceptional. Of twenty-seven cases of subphrenic abscess in connection with gastric ulcer recorded in the post-mortem books of St. George's Hospital, twenty-three were confined to the left side.

Cancer of the stomach rarely gives rise to subphrenic abscess; but owing to the greater liability of the pylorus, if an abscess result it is likely to be upon the right side.

In the case of a simple ulcer the perforation does not always persist. Sometimes it is quite minute and soon stopped by a coating of lymph or by adhesion to some adjacent structure; it may be completely cicatrised by the time the subphrenic mischief comes into operation. But notwithstanding the closure of the perforation the gas increases in volume by fermentative and putrefactive changes.

All the signs of air and fluid in a cavity together are generally present. The coin-sound or bell-note defines accurately the limits of the immediately underlying gas; but tympanitic percussion, on the contrary, is apt to be conducted far up the sternum and over the cardiac area. The fallacious resemblance to pneumothorax is sometimes still further increased by amphoric breathing, though the diaphragm be intact. If the amphoric breathing be of great purity and distinctness, patency of the perforation and passage of air between the stomach and abscess, in accordance with the movements of the diaphragm, probably exist; but no doubt pulmonary sounds may gain something of an amphoric character by transmission through the gas-containing cavity.

Gaseous distension of the stomach is a condition which, so far as physical signs are concerned, may need to be distinguished. In case of doubt, which, however, can rarely arise, the gastric tympanites will be found to have a convex boundary towards the left, corresponding to the fundus of the stomach; while the subphrenic tympanites can probably be traced farther into the axilla, and, under suitable arrangement of posture, even round to the back.

Dyspnœa from compression of the lung is always a prominent symptom. In a right-sided case the dome of the diaphragm may reach the level of the second intercostal space in front: on the left side, owing to the presence of the heart, the displacement is less. In each case the heart is tangibly raised, with little tendency to displacement towards the opposite side: in a left-sided case, indeed, the apex inclines to the same side, owing to the more horizontal position into which the heart is lifted.

Although the contents of the abscess eventually become putrid they are not so at first; thus at first the thoracic complications are of a comparatively innocent kind. Pleurisy is of constant occurrence, and frequently results in complete or partial adhesion of the lung to the diaphragm. There may be effusion of fluid, which, however, is seldom purulent at first, though it subsequently becomes so; and the extent of the empyema is usually limited by adhesions. True pericarditis is rare, though a pleuro-pericardial friction-sound is often present.

Perforation of the diaphragm is a late event, and is most prone to occur near the centre of the wing, where the lung will probably be adherent. Thus the disastrous occurrence of pyopneumothorax may be avoided; but rupture into the lung is scarcely more favourable, and results more often in a gangrenous form of pneumonia than in adequate expectoration of the pus.

Rupture of the abscess in an abdominal direction, when it has once become circumscribed against the peritoneal cavity, is exceptional. Spontaneous evacuation may, however, take place by way of the colon, and even by way of the stomach.

An abscess of the same nature and history as that just described is sometimes found in connection with a simple ulcer of the first part of the duodenum. The peritonitis from perforation of this viscus is far more often diffuse than circumscribed; and if an abscess result, this is likely to be entirely beneath the right lobe of the liver. Adhesions, however, may be so disposed that the greater part of the abscess is immediately beneath the diaphragm, the right wing of which is the one usually occupied. The liver may be greatly depressed; but its position is less oblique than with pneumothorax, and the organ is partially removed from contact with the abdominal wall.

Perforation of the transverse colon is a recognised result, but a rare cause of subphrenic abscess, on either side.

Perforation of the appendix cæci into the peritoneal cavity is an occasional mode of origin. The abscess is mostly on the right side, though seldom connected directly with the original mischief; the probability is rather of a discontinuous chain of peritoneal suppuration along the ascending colon.

The more usual kind of subphrenic abscess from perforation of the appendix and other forms of perityphlitis is mainly retroperitoneal; suppuration in the iliac fossa having spread behind the kidney to the posterior surface of the liver, between the layers of the coronary ligament.

Here it expands into an abscess which, if inflated by gas, covers the right lobe of the liver from behind, and is not in itself easily to be distinguished from the peritoneal variety. It is generally accompanied, however, by inflammatory signs in the lumbar or iliac region.

Retroperitoneal suppuration does not infect the pleural lymphatics; but when of the acute kind just described it has a special tendency to penetrate the diaphragm in front of the external arcuate ligament, where the muscular fibres are more or less deficient. Here the pus often breaks into the pleural cavity before adhesions have had time or opportunity to form; or, less frequently, strips the pleura from its attachments and collects in the mediastinum.

Retroperitoneal suppuration of a more chronic kind, such as perinephritic, is seldom widely subphrenic.

Next in point of frequency to perforation of the alimentary canal come gall-stones as a cause of subphrenic abscess. In most cases the abscess originating in mischief about the gall-bladder occupies the right side; when connected with ulceration of the ducts it is usually upon the left. An irritating quality is often supplied by admixture of bile, leading to perforation into the duodenum, colon, or stomach; and the same quality is displayed in the thoracic complications, among which pericarditis is noticeable.

Hepatic abscess of any kind, making its way upwards, may break into the subphrenic space before the usual adhesions which ensure direct perforation of the diaphragm have been effected. This has often been observed in the case of tropical or dysenteric abscess. Suppuration around a hydatid of the upper or posterior surface of the liver is naturally subphrenic rather than hepatic.

Suppuration in the spleen, so far as it relates to this subject, is usually secondary; but when of embolic origin, it sometimes becomes subphrenic, and perisplenic abscess has been described in connection with malaria.

Blood effused under the diaphragm from laceration of the liver or spleen is liable to infection from the alimentary canal, and to become the seat of suppuration which runs a very rapid course.

Suppuration about the pancreas, or in the lesser peritoneal cavity, is more apt to extend downwards behind the peritoneum than to invade the subphrenium; but when of some duration, and especially when connected with the alimentary canal, it may burrow upwards under the left wing of the diaphragm. Cholelithiasis is the most frequent cause of suppuration in either place, and a secondary connection may be contracted with the stomach, upper part of small intestine, or colon. Moreover, perforation of the posterior surface of the stomach or transverse colon is itself a possible cause of abscess of the lesser peritoneum.

Suppuration about the uterine appendages has been met with as the remote cause of a subphrenic abscess, which, like that from perforation of the appendix cæci, may be either within the peritoneal cavity or behind it.

Any supposed thoracic cause of a subphrenic abscess must be accepted

with caution. Pus may long exist upon the upper surface of the diaphragm without provoking suppuration below. A pleural empyema making its way downwards is far more likely to take the course of a renal or psoas abscess than to accumulate immediately beneath the diaphragm. The latter result has been known to occur; but in most cases where this sequence is suggested it is more probable that the subphrenic was the primary abscess.

The treatment of subphrenic abscess is purely surgical, and it is only necessary here to indicate the broad outlines of it.

The chief object, when once the abscess has become circumscribed and a radical operation is not in question, is to anticipate material complications above the diaphragm; and the only effectual method is by free opening and drainage.

If the abscess be entirely within the bony thorax, it must be opened through the chest-wall like an ordinary empyema; but if it present in the epigastrium there is no doubt of the propriety of direct incision here. This one opening may be sufficient; but, in left-sided cases especially, it is generally advisable to make a counter-opening between the ribs behind. The lower part of the pleural cavity is probably obliterated by adhesions; and in any case the disadvantage of operating through a possibly healthy pleura is outweighed by the paramount importance of free drainage. If this be not secured, the certainty of secondary suppuration in the chest cannot be too strongly insisted upon, or its insidious character too anxiously feared.

W. LEE DICKINSON.

REFERENCES

1. ALLBUTT, T. CLIFFORD. "A Clinical Lecture on Sub-diaphragmatic Abscess," *The Clinical Journal*, 7th June 1893.—2. BARLOW, G. H. "Perforation of the Stomach with Obscure Thoracic Symptoms," *London Medical Gazette*, 1845, vol. i. p. 13.—3. COSSY. "Sur le pneumothorax engendré par des Gaz provenant du tube digestif," *Archives générales de méd.* 1879, t. ii.—4. COUPLAND, SIDNEY. "Subphrenic Abscess simulating Pneumothorax," *Brit. Med. Jour.* 1889, vol. i. p. 636.—5. DEBOVE and RÉMOND. "Des Abscès gazeux sous-diaphragmatiques par perforations des ulcères de l'estomac," *Gazette des hôpitaux*, 1890, p. 1159.—6. EWART. "Abstract of a Clinical Lecture on Cardiac and Hepatic Percussion in the Diagnosis between Pneumothorax, Subphrenic Pneumothorax, and Gastric Gaseous Distension," *The Clinical Journal*, 25th July 1894.—7. FAGGE, HILTON. "Cases of Abscess within the upper part of the Abdomen," *Guy's Hospital Reports*, 1874, 3rd series, vol. xix. p. 213.—8. FENWICK, SAMUEL. *Clinical Lectures on some obscure Diseases of the Abdomen.* London, 1889.—9. JENDRÁSSIK. "Ein Fall von subphrenischen Abscess. Beitrag zur Erklarung des Zwerchfellphänomens," *Deutsche med. Wochenschr.* 1895, No. 40.—10. LEITH. "Inflammatory Lesions of the Thoracic Contents arising from Subdiaphragmatic Conditions due to Gastric Ulcer," *Edinburgh Hospital Reports*, 1895, vol. iii.—11. LEITH. "Intrathoracic Inflammation following upon Subdiaphragmatic Suppurations, arising from Lesions of the Liver, Spleen, Kidney, Intestine, and Vermiform Appendix," *ibid.*—12. LEYDEN. "Ueber Pyopneumothorax Subphrenicus und Subphrenische Abscesse," *Zeitschr. f. klin. Medicin*, 1880, Bd. i.—13. LEYDEN and RENVERS. "Ueber Pyopneumothorax Subphrenicus und dessen Behandlung," *Berlin klin. Woch.* 1892, No. 46.—14. LITTEN. "Das Zwerchfellphänomen und seine Bedeutung für die klinische Medicin," *Wiener klin. Wochenschr.* 1895, No. 5.—15. MASON. "Subphrenic Abscess, with special Reference to those Cases which simulate Pyopneumothorax,"

Trans. Assoc. Amer. Phys. 1893.—16. MAYDL. *Ueber Subphrenische Abscesse.* Wien, 1894.—17. NOWAK. "Die hypophrenischen Empyeme," *Schmidt's Jahrbücher*, 1891, Bd. 232, S. 73 and 200.—18. PENROSE and LEE DICKINSON. "Cases of Abscess beneath the Diaphragm in connection with Perforating Gastric Ulcer," *Clin. Soc. Trans.* 1893, vol. xxvi.—19. SÄNGER. "Drei Fälle pneumo-perforativer Peritonitis, nebst Bemerkungen über Subperitonitis und Pleuraperforationen," *Archiv für Heilkunde*, 1878.—20. SCHEURLEN. "Ueber Pyothorax Subphrenicus," *Charité-Annalen*, 1889.—21. TAYLOR, FREDERICK. "A Case of Abscess between the Diaphragm and the Liver," *Guy's Hospital Reports*, 1874, 3rd series, vol. xix. p. 257.

W. L. D.

DIAPHRAGMATIC HERNIA

PHRENIC or diaphragmatic hernia is a protrusion of part of the abdominal viscera into the chest, and usually takes place through a gap in the diaphragm which is either a congenital deficiency or the result of injury, especially rupture from indirect violence. The hernia is very rarely contained in a sac: occasionally, however, a sac is provided by a congenital pouch of the diaphragm, or forms gradually in elderly persons at some weak spot in the muscle, such as that which normally exists at the side of the ensiform cartilage. As met with clinically, the hernia itself is seldom congenital; not that this condition is relatively uncommon, but that for the most part it interferes fatally with the proper establishment of respiration at birth.

In the great majority of the cases which present themselves in practice the hernia is of sudden onset, and can be traced to some definite strain or fit of abdominal distension, if not to obvious violence. Dr. Clifford Allbutt tells me of a case under his own observation in which left diaphragmatic hernia, suddenly established, ended the life of a man who, at the great age of eighty-two, had reached convalescence after an unmistakable and by no means mild attack of enteric fever.

With about equal frequency the lesion in the diaphragm is congenital or traumatic. Congenital gaps are of various extent, and may amount to complete absence of one wing of the diaphragm; but for practical purposes they are restricted to a deficiency of the muscular part posteriorly, through which the peritoneal and pleural cavities communicate. They occur chiefly on the left side. Ruptures, also, though by no means constant in their disposition, are apt to be chiefly posterior. On the right side the diaphragm is in great degree protected from rupture by the liver, which organ, moreover, serves as a guard to any existing gap; consequently the hernia is nearly five times more common on the left.

The organs most frequently implicated are the stomach and transverse colon; and after these the more movable parts of small intestine, the spleen, and the left lobe of the liver. Even the duodenum and pancreas may be dragged in.

With a view to surgical treatment exact diagnosis is of the greatest importance, but this is too often impossible. However, the physical signs of a large protrusion through the diaphragm, consisting mainly of stomach

and intestines, are sufficiently characteristic. The abdomen falls in, while the affected side of the chest is filled out and the respiratory sounds are entirely suppressed over a considerable area, the greater part of which is probably tympanitic on percussion. The heart is pushed far to the opposite side. The thoracic signs, in fact, are not unlike those of pneumothorax, for which they would be easily mistaken were it not for the state of the belly. Such cases generally depend upon free laceration of the diaphragm, and for the time there may be little or no obstruction of the herniated organs. The leading symptom is dyspnœa, though it is not likely that vomiting will be absent. Partial recovery is not unusual; but, sooner or later, perhaps not till after many years, strangulation of some part which remains in the chest is almost invariably the immediate cause of death.

In the case of a congenital gap the hernia is apt to become strangulated early. Urgent vomiting sets in at once, only to aggravate the mischief; and the entire stomach may be forced into the pleural cavity. Under these circumstances the greater curvature of the stomach is uppermost and the posterior surface in front; and the dislocation is sometimes so sharp that the opening of the œsophagus is obstructed.

Nothing can be usefully said about treatment except as regards relief by surgical operation. The scanty experience at hand tends to indicate that, with a sure diagnosis and no special reason for opening the abdomen, it is on the whole more hopeful to operate through the chest. The reduction of the hernia, at any rate, is certainly facilitated by letting air into the pleural cavity; and this may be done the more boldly if pneumothorax has already existed in effect.

W. LEE DICKINSON.

REFERENCES

1. ABEL. "Ein Fall von angeborenem linkseitigen Zwerchfellsdefect," etc., *Berlin. klin. Woch.* 1894, Nos. 4 and 5. — 2. DUGUET. *De la Hernie diaphragmatique congénitale.* Paris, 1866.—3. JAFFE. "Case of true Congenital Diaphragmatic Hernia or Pouch," *Trans. Path. Soc.* xlv. 1894.—4. LACHER. "Ueber Zwerchfellshernien," *Deut. Arch. f. klin. Med.* xxvii. 1880.—5. PAGET, STEPHEN. *The Surgery of the Chest.* London, 1896.—6. PEACOCK. "Case of Diaphragmatic Hernia," *Trans. Path. Soc.* xiv. 1863.—7. WINKFIELD. "Supposed Passage of Stomach into Pleural Cavity," etc., *Lancet*, 23rd January 1897.

W. L. D.

ABDOMINAL DIAGNOSIS FROM A GYNÆCOLOGICAL STANDPOINT

THE subject of gynæcological diagnosis is a very large one; it has received special consideration in the companion *System of Gynæcology*, and to that volume the reader is referred for a complete exposition of the many important topics it comprises.[1] The object of the present article is to consider some of the difficulties which the general physician may meet with in investigating morbid conditions of the abdomen and pelvis, and to assist him in excluding those which fall more directly within the province of the gynæcologist. Its purpose is, therefore, a very limited one. No attempt will be made to describe the various difficulties the gynæcologist may have to meet in his own special work, but rather to give an outline, necessarily concise and imperfect, of those diseases of the feminine reproductive organs and their surroundings which are to be excluded from an investigation made from an exclusively medical point of view.

In examining the abdomen, the physician will meet now with one now with another actual or apparent growth, tumour, or enlargement; and he has to decide the character and relations of each, either by his own methods of physical diagnosis, or possibly, in some obscure cases, by means of the assistance of the surgeon or gynæcologist, who may clear up the question in doubt by some such operative procedure as exploratory puncture or laparotomy.

For the purposes of the present article we have to consider such conditions only as are primarily abdominal, but even in these cases vaginal examination may sometimes be necessary to clear up doubtful points; conditions which are essentially pelvic, and which are to be made out by vaginal examination only, do not fall within my subject.

The various forms of abdominal tumour connected with the reproductive organs of women first claim attention, and the physician has to decide whether an apparent abdominal enlargement be really caused by a definite localised growth or tumour; and, if so, whether such tumour is one falling within his own province or within that of the gynæcologist.

To make a satisfactory examination of the abdomen it is necessary

[1] See *System of Gynæcology*, art. "Diagnosis in Gynæcology," by Robert Boxall, M.D.

that the patient should be in bed, undressed, with her shoulders slightly raised, the surface of the abdomen uncovered, and the thighs flexed so as to relax the abdominal walls. No false notions of modesty should induce the physician to be content with any such partial and imperfect investigation as is often attempted by palpation when the patient is clothed, or when the surface of the abdomen is covered even though it be by a single fold of linen. Any such method is defective in accuracy of observation, and in some cases may lead to serious error.

Mere inspection, as a rule, will not afford much useful information. In occasional cases of extreme flatulent distension, simulating tumour, the peristaltic movements of the small intestines may be observed on the surface.

More important information can be obtained by palpation, which is of paramount importance in deciding the nature and relations of many intra-abdominal enlargements.

In practising abdominal palpation care is needful. One of the chief obstacles to its successful application is involuntary contraction of the muscles, which renders the abdominal walls so hard and rigid that satisfactory palpation becomes quite impossible. To remonstrate with the patient is useless; the more her attention is directed to the matter the more nervous and apprehensive she is apt to become, so that the muscles of the abdomen become as hard as a piece of wood. It is best to enter into conversation with her on other matters, keeping the hands on the abdomen until her attention is diverted from the examination in progress. Sometimes when the abdominal contents cannot be felt at all, the patient may be directed to hold her breath as long as possible. Eventually she will be obliged to take a deep inspiration; at the conclusion of this her abdominal muscles will relax, and then the existence of any tumour can be made out.

When accurate examination of the abdominal contents is impossible by reason of rigidity of the muscles, it is better to examine the patient under an anæsthetic. Then any questionable growth can be freely palpated, percussed, or moved about without hindrance. In certain cases in which an enlargement is simulated by muscular contraction and abdominal distension, as in the so-called phantom tumour or spurious pregnancy, this simple expedient will clear up the nature of the case at once, the projection of the abdomen disappearing as narcosis is established, and reappearing as soon as the patient regains her consciousness.

In practising palpation of the abdomen the palms of both hands should be laid flat on the integuments, and then rotated gently towards their ulnar margins, so that the surfaces below them may be felt. Many persons defeat this object by pressing downwards with the tips of the fingers, which generally produces further contraction of the muscles.

Next to palpation percussion is the most valuable means of acquiring information as to abdominal tumours. The percussion note over a tumour of the uterus or ovaries is dull, and, with rare exceptions, extends downwards to the pelvic brim. The intestines are pushed upwards and

to either flank by such growths, and resonance therefore surrounds them. If there is a dull note on percussion in the flanks but not in the centre of the abdomen, or if there is a clear and distinct area of resonance between the dulness and the pelvic brim, then this dulness is presumably caused by something which does not come within the province of the gynæcologist. It will be well, however, to bear in mind the possibility of a coil of intestine being adherent to the surface of an underlying ovarian or uterine growth, and thus masking the presence of the latter by an area of resonance.

Auscultation is chiefly useful in distinguishing an abdominal growth due to pregnancy from other forms of tumour. With ordinary care the fœtal heart sounds can hardly be mistaken for anything else, and by this means a doubtful case can be cleared up. Other sounds are occasionally audible in abdominal tumours of a pelvic origin, such as vascular souffles in fibromyoma, or the transmitted pulsations of the aorta; but from a diagnostic point of view they are of little practical value.

In abdominal tumours of an elastic or fluctuating character an exploratory puncture by an aspirating trocar is often practised to clear up the diagnosis; but this practice was much more frequent formerly than at the present time. As a matter of fact, there are few cases in which this procedure is essential or even advisable from a diagnostic point of view; and it is one which is always attended with the risk of septic infection. If practised at all the most rigid antiseptic precautions should be adopted, both in the cleansing of the skin at the point of puncture, and in the thorough purification of the exploring needle.

In examining the abdomen under conditions which are properly obstetric or gynæcological, the first physical characteristic calling for consideration is the tumour caused by normal pregnancy. The reader who is unaware that even the most experienced abdominal surgeons have fallen into error either by overlooking the existence of pregnancy altogether, or by mistaking it for some other condition, may be disposed to underrate the difficulties occasionally met with in arriving at an accurate decision. One peculiarity of the tumour formed by pregnancy is that it may vary greatly in dimensions. We may meet with the small ovoid only just above the pelvic brim, readily to be overlooked in stout women with rigid abdominal walls, which indicates a pregnancy of from three to four months' duration; or we may have a large tumour almost completely filling the abdomen, indicating a pregnancy nearly at term, or again a mass of any intermediate size. Whatever its size the tumour of pregnancy is of an ovoid form, its longest diameter corresponding with the long diameter of the abdominal cavity; or, in advanced pregnancy with a transverse position of the fœtus, the long diameter of the tumour may lie obliquely across the abdominal cavity: too much reliance, however, must not be placed on these relations, as it is far from uncommon to find the pregnant uterus lying decidedly to one side or the other. On palpation it is uniform in outline, and freely mobile from one side of the abdomen to the other. It must not be forgotten that

pregnancy may have occurred in a uterus in which there were already one or more fibromyomatous growths; so that the presence of the characteristic hard nodular masses of such tumours does not necessarily exclude pregnancy, the presence or absence of which must be settled by other means. In pregnancy there is an elasticity to the touch, different on the one hand from the hardness of most uterine tumours, and on the other from the fluctuation of ovarian cystoma.

The most characteristic point, however, is that the density of the pregnant uterus is variable. After the uterus is sufficiently large to be felt by palpation, if the hand be placed over it, and it be grasped for a time without using any friction or pressure, it will be felt to harden distinctly, in a manner that is quite characteristic. This intermittent contraction occurs every five or ten minutes, sometimes oftener, rarely at longer intervals. This is unquestionably a sign of great value in differential diagnosis. If the hand be kept steadily on the uterus, its alternate hardening and contraction can be made out with the greatest ease. Physicians are apt to rely on the movements of the fœtus felt through the uterine walls as a sign that an abdominal tumour is a pregnant uterus; these movements, however, are not perceptible till the period of pregnancy is far advanced, and they are apt to be simulated by other conditions, such as semi-involuntary contraction of the abdominal muscles, and movements of the intestines distended with flatus. But the alternate relaxations and contractions to which I have just referred are normal accompaniments of gestation; they may be felt at any period of pregnancy, and they are beyond the control of the patient. Thus they form a ready and trustworthy means of distinguishing the gravid uterus from other abdominal enlargements.

Whenever the tumour reaches up to or above the umbilicus auscultation may assist us. The uterine souffle, although it may be heard in uterine tumours, will not occur in abdominal enlargements which are not "gynæcological"; and its presence may, therefore, be sufficient of itself to show that the tumour under examination does not fall within the province of the physician. The sounds of the fœtal heart will, of course, clear up the diagnosis beyond any possibility of error. The converse, however, is not to be relied upon; cases of advanced pregnancy are very common in which, for some reason or another, neither the uterine souffle nor the fœtal heart can be heard.

The corroborative signs to be found elsewhere must not be overlooked; of these the most trustworthy are enlargement of the breasts, pigmentary changes in the areolæ, and the presence of lacteal secretion.

By the vagina the softened cervix, the enlarged uterine body—to be made out by bimanual examination, and the existence of ballotment—are very characteristic.

Distension of the uterus by retained menstrual fluid or watery secretion is an occurrence so rare that it can seldom give rise to error. Still it occasionally happens that the uterus becomes enlarged in this way; sometimes it may reach even to the level of the umbilicus, and the physical

character of such a tumour is not unlike that of the gravid uterus. The best safeguard against mistake is the previous history of the case, which will always be different from that of ordinary pregnancy; although there may possibly have been attacks of pain, pyrexia, general wasting, or constitutional disturbance, which may mislead the physician. Retention of menses is always the result of some physical obstruction to the exit of the fluid, such as imperforate hymen; or if it occur in women who have already menstruated, we may usually trace a history of some cause, such as inflammation following an antecedent labour, which has led to occlusion of some parts of the genital tract. The existence of a pelvic or abdominal tumour in a girl who has never menstruated will of itself give rise to suspicion, as pregnancy under such circumstances is extremely uncommon. Whenever suspicion of the true nature of such a case has arisen, a careful vaginal examination will generally clear it up. The obstruction is generally in the vagina, and is at once detected; the vaginal canal above it is felt through the rectum to be greatly distended by fluid; and we may also find a bulging and imperforate hymen protruding through the vulva. The absence of mammary changes, and of ballotment, will materially aid us in forming a diagnosis.

The physician may occasionally have to deal with certain very severe and obscure cases, the result of an extra-uterine fœtation; he may then be very liable to fall into error. The intense cataclysmic shock resulting from a rupture of a tubal fœtation might well be referred to some other intra-abdominal condition; the more so as it often occurs suddenly without any previous recognition of pregnancy. The fact that in most cases of the kind there has been an irregular hæmorrhagic discharge from the uterus, which has been mistaken for menstruation, is also very apt to mislead. In cases in which the patient does not at once succumb, a secondary intense peritonitis is apt to supervene and may still farther obscure the diagnosis. Error can only be avoided by a careful investigation of the history of the case. Such an investigation will probably show that some symptoms, however obscure, of early pregnancy existed; and vaginal examination may then clear up the case by showing the presence of a tumour in the position of one or other Fallopian tube, or the presence of effused blood extravasated in the neighbourhood of the uterus, especially if the tube has not ruptured into the general peritoneal cavity.

In the so-called abdominal pregnancy, that is in cases in which the fœtus did not perish at the time of rupture of a tubal gestation but became encysted in the abdominal cavity, the diagnosis from other abdominal conditions is often extremely difficult, or even impossible. The difficulties, however, are those which the gynæcologist has to deal with rather than the general physician. The exact nature of the resulting abdominal enlargement may be a matter of extreme doubt, but the history of the case will probably show clearly enough that the state of the patient is caused by some morbid state of the reproductive organs. The tumour will generally be found closely adherent to the uterus, and may resemble a uterine growth; but, generally speaking, the signs of peritonitis,

fixation of the growth to the surrounding structures, and pelvic deposit, can be made out per vaginam.

The uterine tumours, unconnected with pregnancy, which are most frequently met with are the various fibromyomata. The majority of them are readily distinguished from other abdominal growths. The most common kind are hard, nodular, painless and more or less mobile; and by combined vaginal and abdominal examination their connection with the uterus can easily be made out. If, however, fibromyomata be loosely attached by a pedicle to the walls of the uterus, they may be so free in the abdominal cavity as to be hardly distinguishable from solid growths of the omentum or peritoneum; they have even been confounded with a movable kidney or with the spleen. By careful palpation we may feel the uterine attachments, while the peculiar shape of the kidney and spleen will generally enable us to distinguish these viscera. It is certain, however, that enormous enlargements either of the kidney or of the spleen are occasionally met with which may closely resemble solid tumours of the uterus. Not long since I saw a huge abdominal growth, nearly filling the abdomen, of which a large lobular portion was felt per vaginam in the right broad ligament; the case had been seen by several most experienced abdominal surgeons, and diagnosed by all of them to be uterine. Laparotomy was performed, and on opening the abdomen the tumour was found to be the spleen, which had grown to such a size that it not only filled the abdominal cavity, but had burrowed deeply into the pelvis on its right side, displacing the uterus towards the left ileum. In such an exceptional case error was inevitable. Such a case is different from the so-called "wandering spleen," in which the viscus is found in the lower abdomen. In these rare cases the peculiar shape, the splenic notch, and the absence of splenic dulness in its proper place, may enable us to form a correct diagnosis. The fact that surgeons of great experience, proposing to perform hysterectomy, have found on opening the abdomen that the tumour concerned was renal, shows that diagnosis in such cases is not always an easy matter. Probably error may be best avoided by tracing the tumour into the pelvic cavity, both by palpation and percussion, and, in the case of suspected uterine growths, by ascertaining with the uterine sound whether the uterine cavity is or is not elongated. Tumours of the ovary felt in the abdominal cavity are generally cystic, and are distinguishable by their fluctuation on palpation. Too much reliance, however, must not be placed on this sign, since in multilocular ovarian tumours, with viscid contents, the impression given will often be that of bogginess or elasticity rather than of definite fluctuation. When the cyst walls are thin, and the contents of the tumour are not viscid, the impulse may be so readily made out as to suggest the presence of free fluid in the peritoneal cavity. In either case we may be able to settle the point by vaginal examination, when we may possibly feel portions of the tumour in the pelvis, or be able to make out its attachments.

Error can be avoided in most cases by percussion, since in ovarian tumour the percussion note is uniformly dull over the site of the tumour,

with a surrounding area of resonance from the intestines which are displaced all round it; whereas in ascites there is resonance over the surface and dulness in the flanks, the area of resonance being, moreover, altered by changes in the position of the patient, the free fluid gravitating into the most dependent portion of the peritoneal cavity.

The variety of tumour most likely to be mistaken for free fluid in the peritoneum is the parovarian cyst which, being thin-walled and unilocular, is very fluctuating, and is often difficult to recognise. Here again the situation of the dulness over the surface, the fact that the area of dulness does not shift by change of position, and the possibility of feeling the cyst by combined vaginal and abdominal examination, will be the best means of avoiding error.

Solid growths of the ovary are rare, and are more apt to be confounded with uterine tumour than with conditions likely to come before the physician. Generally they are comparatively mobile and unilateral, and their nature may be verified by vaginal examination.

Extreme distension of the bladder by urine may give rise to an abdominal tumour which is apt to be deceptive, since it may be accompanied by great abdominal pain, and probably by some pyrexia; and thus may be taken for peritonitis, or some other intra-abdominal condition. The tumour itself so formed may readily escape observation, or it may be taken for a uterine or ovarian growth. I have seen such a case mistaken for one of ovarian tumour; in it the distended bladder rose a long way above the umbilicus, and its bursting was prevented only by the dribbling of urine from the urethra. Of course the passing of the catheter will at once clear up any doubtful case of this kind, a simple precaution which should always be taken when we are investigating the nature of an obscure tumour.

Tumours connected with the Fallopian tubes, such as pyosalpinx, hydrosalpinx, or hæmatosalpinx, or tubular fœtation, although very important from a gynæcological point of view, are little apt to mislead the general physician, as they are seldom large enough to be felt in the abdomen. A large pyosalpinx on the right side, however, accompanied by severe pain and pyrexia, and cropping up above the pelvic brim, might readily enough be confounded with appendicitis; but such an error would be avoided in all doubtful cases by a vaginal examination, which will enable us to feel the distended tube through the right vaginal fornix.

There are a considerable number of cases in which swellings of a gynæcological origin, often of considerable size, are found in the lower abdomen, and might readily lead to errors of diagnosis. Thus in certain cases of pelvic peritonitis the adhesions between the intestines, and the exudation resulting from the inflammation, may form a mass of considerable size which might readily enough be mistaken for an omental tumour or malignant disease of the peritoneum. The possibility of such a mistake was impressed on my mind by a strange case which I saw in the first year of my practice in London. I was present, for the purpose of administering an anæsthetic, at a consultation

held between four of the most eminent members of the profession, on the case of a young lady who had been brought up from the country for their opinion. She was to the last degree emaciated and cachectic, she had been confined to bed for many months by some obscure abdominal condition, and she had a large oval, hard, smooth outlined tumour, reaching midway between the umbilicus and the ensiform cartilage. The unanimous conclusion was that it was a mass of malignant disease; and I have a vivid remembrance of the painful scene that followed on the announcement of this fatal verdict, when the husband fell to the ground in a swoon. The patient was taken home to die, and in the railway carriage, probably from the jolting, the tumour ruptured at the umbilicus, a large quantity of pus was discharged, and a rapid and complete recovery followed. This occurred before the days of the clinical thermometer, and the temperature chart, showing the pyrexia which would usually accompany such a case would now render such an error impossible. Moreover, in cases of this kind the tumour could be traced into the pelvis, and then we could ascertain its true character by vaginal examination, which would show complete fixation of the uterus and considerable periuterine inflammatory deposit.

Swellings caused by pelvic cellulitis or parametritis are hardly likely to be mistaken for other forms of abdominal disease. They are generally more or less lateral, and occupy one or other iliac fossa. The fact that they are at times singularly painless, and that they are dense and hard on palpation, might lead us to conclude that they were not inflammatory in origin; and this impression might be strengthened by the fact that sometimes the pelvic cavity, as ascertained by vaginal examination, does not contain any deposit or exudation. The clinical history should prevent such an error, since such forms of inflammation are generally puerperal, and there is a distinct pyrexial temperature.

Other abdominal swellings of a somewhat analogous character to those resulting from pelvic peritonitis may be caused by effusions of blood. Here again the clinical history should prevent errors in diagnosis, since there is always a sudden illness at the time when the blood is effused, and there are practically always vaginal deposits in the periuterine tissues, with fixing and displacements of the uterus itself, which should clearly indicate the character of the abdominal swelling.

W. S. PLAYFAIR.

ENTEROPTOSIS

THE names enteroptosis or visceroptosis have been applied to cases in which various abdominal organs have become displaced from their normal positions, which displacements are practically all in the downward direction. The "dropping of the viscera" is an intelligible English equivalent for these names. The thing signified is rather a general dropping or prolapse of all the movable abdominal organs than a disturbance in the position of an isolated viscus. With visceroptosis certain symptoms are associated which may or may not be directly dependent upon the apparent anatomical lesion.

It will be convenient, first, to consider the manner in which the chief viscera are suspended or supported; next, to deal—in the briefest manner—with displacement of the several viscera; and, finally, to discuss general ptosis of the abdominal organs, and that medley of symptoms which has been described under the title of Glénard's Disease.

1. Anatomical considerations.—The abdominal viscera are held in place by certain folds or stretches of peritoneum on the one hand, and on the other by certain adhesions here and there between such viscera and the retro-peritoneal connective tissue. The blood-vessels which pass to an organ may take some share in maintaining it in position, as indeed may any other structure with which it is directly connected. The *stomach*, for example, is suspended by the œsophagus, and further, its cardiac extremity is held in place by a reflexion of the peritoneum to the diaphragm (the gastro-phrenic ligament). Beyond the point of entrance of the gullet the stomach is maintained in its transverse or oblique position by the lesser omentum, which attaches it to the liver, and by its blood-vessels, more especially by those which derive from the hepatic artery. So far as the maintenance of its position is concerned, the great omentum which passes to the transverse colon, and the gastro-splenic omentum which passes to the spleen, are means of possible disturbance. The *liver*, owing to its weight and its relation to the ever moving diaphragm, needs to be substantially supported. This need is met by the inclusion of the organ between the layers of the gastro-hepatic omentum. By means of this omentum the liver is firmly attached to the diaphragm, and by means of the round ligament it obtains a substantial hold of the anterior abdominal parietes. The *spleen* is entirely

surrounded by peritoneum, except at the hilum, and is slung by folds of peritoneum between the stomach and the diaphragm. Its position is maintained in some degree by its vessels, and by its close association with the kidney. One would have supposed that of all the organs in the abdomen the spleen was the least fitted to maintain a constant position. Its displacement, however, is quite uncommon. The *small intestines*, being suspended from the spinal column by the copious mesentery and the great mesenteric vessels, are well attached. The ascending and descending portions of the *colon* owe the security of their position to the shortness or absence of the mesocolon; the sigmoid flexure lies about the brim, or within the cavity of the pelvis, while the transverse colon is held in precarious position by the unstable transverse mesocolon. This part of the great intestine is burdened by the great omentum on the one hand, and supported by the flexures of the colon on the other. The *kidney* is held in place by its capsule of fat-bearing connective tissue. A partial covering of peritoneum is loosely adherent to its anterior surface. Lodged as it is at the back of the abdominal cavity one would have thought that its position would seldom be disturbed, and that at least it would be infinitely less prone to wander than the spleen. Clinical experience, however, shows that the kidney is of all solid organs in the abdomen the one most disposed to break from its attachments. [*Vide* art. "Floating Kidney" in the next volume.]

2. **Displacements of the viscera severally.**—On examination of the abdomen, and especially in the course of the inspection which is permitted by exploratory incision into the cavity, displacements of individual viscera are discovered which, on the one hand, may be quite unexpected, and, on the other, may give rise to misleading symptoms.

The Stomach.—It would appear that the healthy stomach is very seldom displaced; but when it is the seat of any growth, and especially when dilated from obstruction at the pylorus, it may be very considerably disturbed in its space relations. This subject is fully dealt with in the article on "Dilated Stomach" (p. 490, Fig. 21). I have found the healthy stomach considerably below its normal level; even with its greater curvature on a line with the iliac crests. The viscus in such cases is usually displaced to the left rather than to the right, and the pylorus drops well to the left of the median line. The stomach—presumably normal—has been opened by error in the performance of a left lumbar colotomy. The "vertical stomach" described by some authors must, I think, be exceedingly rare. I have never met with an instance of it in surgery. Certain of the displacements just described occur in the subjects of gross spinal deformity, others occur in persons who are normal as regards the skeleton; and not a few are met with in thin women who exhibit the conformation of the lower part of the thorax which is rightly or wrongly ascribed to tight lacing. Rüdinger gives an excellent representation of this deformity; the subject was an unmarried woman of twenty-two, and the distortion is ascribed by him to the wearing of tight corsets. In such

persons the lower part of the thorax is much narrowed, and there the transverse section of the thoracic cone is smallest: thus the abdominal aperture of the thorax is much constricted; the costal margins below the xiphoid cartilage are, for a long way, parallel to one another, and are separated by quite a narrow interval. The thorax appears to be unduly long and narrow, and the patients describe themselves as "long waisted." I doubt whether this condition is always due to tight lacing; I have met with it mostly in women of middle age, and in not a few in whom the evidence of undue constriction of the waist was entirely lacking. Yet in one of these persons I found the stomach, otherwise quite normal, extending well into the left iliac fossa.

I am not aware that these displacements of the healthy stomach give rise to any conspicuous symptoms, nor is the condition easily recognised except on abdominal operation. The instances which have come under my notice have been discovered unexpectedly in this way.

The Liver.—The normal liver may be remarkably displaced in the subjects of spinal deformity, and in women who present the elongated and constricted thorax above described. In two instances in such patients the displaced liver had been mistaken in one case for a tumour, and in the other for hypertrophy of the liver. In both examples the margin of the liver lay some way below the umbilicus, and the mobility and prominence of the organ—the patients being thin—were remarkable. Examination showed that the organ was normal both in size and shape. The patients were dyspeptic and feeble in health, and their intellectual life centred around the supposititious abdominal tumour. The actual disturbance produced appeared, however, to be trifling. In both instances the displacement had not been recognised until the patients had passed middle life.

The Spleen is apt to become considerably displaced, and the wandering spleen has given rise to many erroneous diagnoses. The wandering spleen may be found in any part of the abdomen, and may change its position frequently and mysteriously. It may lodge in the right iliac fossa, or may pass down into the pelvis and become manifest on a vaginal examination (*vide* p. 584). This exceptional degree of mobility is rarely met with except in women, and is found for the most part in those who have borne children.

The spleen may be thrust entirely out of place in certain subjects of extensive spinal deformity; but it need not thus become unduly movable. A spleen so displaced may be mistaken for a new growth in the omentum. In persons with the constricted thorax, described in speaking of the stomach, the spleen may be considerably displaced downwards, but may not be movable.

The true wandering spleen may give rise to much inconvenience. It may roll about the abdomen like a foreign body. The pedicle may become twisted, and acute symptoms be produced akin to those which attend the twisting of the pedicle of an ovarian tumour. Such axial rotation may be followed by atrophy of the whole organ or, in acute

examples, by gangrene. Independently of rotation the wandering spleen may become so engorged with blood as to be more than doubled in size. The greatly engorged organ may be readily ruptured by a moderate degree of violence. The wandering spleen may contract adhesions in an abnormal situation. By dragging upon the stomach it may lead to dilatation of that viscus, to acute dyspepsia, to cramp-like pains and to vomiting. By dragging upon the duodenum it may cause compression of the common bile-duct, and so lead to repeated attacks of jaundice. The pancreas may be drawn out of place, and its tail may be found as a thin strip of tissue in the splenic pedicle (Rokitansky). The wandering spleen has caused intestinal obstruction, has led to acute retroflexion of the uterus, and even to prolapse of that organ.

It is no matter of wonder that the wandering spleen has been mistaken for ovarian, renal, and other tumours, and has been the origin and the focus of complex neurotic symptoms.

The only treatment for the wandering spleen, when it gives trouble, is by operation. An attempt may be made to fix it in place by suturing, although this measure has not as yet been attended by much success. Failing this the troublesome viscus should be removed.

The Intestines.—A prolapse of the entire mesentery has been repeatedly noted. In such cases the suspensory tissues at the root of the mesentery appear to yield, and the whole structure glides downwards over the lumbar spine, the peritoneum becoming attenuated, no doubt, and the mesenteric vessels lengthened. It has been urged that prolapse of the mesentery plays an important part in the production of hernia. With this ptosis of the mass of the small intestine is associated some prolapse of the flexures of the colon and often of the kidneys. The condition is a feature of age, and mostly of advanced age. It seems to be most common—as may be surmised—in the feeble, in those of relaxed tissue, and in those who have taken but little exercise. It is often seen in association with the bowed back.

It is asserted that this prolapse gives to the abdomen a distinct conformation, the parietes above the umbilicus being sunken in and flattened while the belly below is bulging, flabby, and abruptly pendulous. The subjects of this not uncommon peculiarity in the contour of the abdomen are as often men as women, and are always thin and spare persons. Dyspepsia, flatulence, constipation, and dragging sensations appear occasionally to be associated with this condition, but by no means invariably.

Of the several parts of the intestine the transverse colon is the segment which is most often found out of place. This displacement is frequently due to some adhesion contracted by the omentum, or to an abiding distension of the colon depending upon chronic constipation. The summit of the transverse colon may be found in the pelvis, and I have many times encountered it in performing left inguinal colotomy, or in exposing the vermiform appendix in the right iliac fossa. It is bent into the form of a V or a U, and may be fixed in that position. Such distortion

must aggravate any existing condition of constipation. The cæcum may be found deep in the pelvis or displaced to the left of the median line; or it may lodge in the left iliac fossa.

The subject of *movable kidney* will be dealt with in the next volume.

3. General ptosis of the abdominal viscera.—*Glénard's disease.*—There is a condition in which a general ptosis or general downward displacement or dropping of the more movable viscera has occurred. With this condition—or at least with some phases of it—certain nervous phenomena are associated; the precise nature of this association and the interpretation of its conditions are very uncertain.

Abdominal ptosis, visceroptosis, or Glénard's disease, has attracted attention during the last ten years only. Glénard's monographs appeared in 1885 and 1886 (1).

An excellent account of the condition is given by Grasset and Rauzier. The disease depends, in the main, upon a relaxation of the abdominal wall and of the supporting ligaments of the viscera, as a result of which the heavier organs drop to a lower level in the abdomen. The affection is usually met with in women, and mostly in young women who are thin and feeble. No definite causes have been assigned to it, although some attribute it to repeated pregnancies, to undue exertion, and to injuries.

It is stated that the right bend of the transverse colon is the first part to descend; the stomach is then so drawn down that the pyloric opening is compressed and the passage of food hindered. In due course the rest of the transverse colon descends, the jejuno-duodenal orifice is narrowed, and a further obstruction is offered to the passage of alimentary matters. The whole mass of the small intestine becomes prolapsed, the lower part of the abdomen is prominent, flabby, and possibly pendulous, while the upper part is flattened. The liver and kidney become loose, and are described as "floating." Certain it is that the kidney becomes movable, and the liver is found to occupy an unduly low level.

The results of this general ptosis may be manifold and distressing. These are, in the first place, certain asthenic symptoms, general depression, and ill-health. The patient becomes an invalid, and is unfit for any exertion. She is readily tired, and is very liable to fainting, and is only comfortable when lying down. There is a sense of "weight" in the abdomen, and of a sickening "dragging." There is pain in the back, and a continued sense of weariness. The patient complains of a "hollow over the stomach," and is often troubled by the pulsations of the aorta, which are unduly evident. Gastric symptoms are prominent; the most conspicuous are a sense of burning in the epigastric region, vomiting, pain, loss of appetite, distress after food, and more or less definite dyspepsia. The vomited matter is often very acid, and if it run over the chin irritates and reddens it. The bowels are irregular; there may be diarrhœa, but more usually there is constipation; the movements of the bowels are attended with pain, aperients cause distress, and relief has to be sought by means of enemas. The stomach and intestines are very apt

to be found dilated; the colon is conspicuous; pressure upon the bladder may be complained of. The symptoms usually accredited to movable kidney may be present. Colic is common, and now and then may be acute.

One important feature remains to be noticed; namely, that the symptoms are more or less relieved by pressing upon the lower part of the abdomen with the two hands, or by the wearing of a supporting belt. Many patients are unable to move about until they have adjusted their supports or bands.

The physical signs need not be dwelt upon. The movable kidneys and the prolapsed liver and stomach return more or less to their normal position when the patient is recumbent.

The examination of the abdomen should always be made while the patient is standing erect. The position of the viscera, and especially of the liver, is ascertained first of all while the patient is in this attitude, and is then noted again when the patient is lying down on the back. The symptoms above described are liable to considerable variation. In many cases vomiting and pain are the leading features. The vomiting is quite inexplicable, and the patient is generally found to have adopted some remarkable plan of diet. The position of the pain varies; but I have been much struck by the frequency with which persisting pain and tenderness are complained of at one definite small spot a little to the left of the median line and just above the level of the umbilicus. This spot would roughly correspond to the inferior mesenteric vessels and nerves. The patient will often cry out when this spot is pressed upon, and the precision with which it is localised is remarkable. I have noticed the same localisation of pain and tenderness in those unhappy people who may be called "intestinal hypochondriacs."

The further peculiarities and the general bearings of abdominal ptosis may be illustrated by the three well-marked cases which follow:—

1. An unmarried lady of fifty-two consulted a medical man about a "tumour" which she had discovered in her abdomen, and which was only to be felt when she was standing up. She was a thin woman of feeble muscular development, who led a very uneventful and sleepy life. The climacteric had been reached some years previously.

I examined the abdomen when the patient was erect and found all the viscera prolapsed. The "tumour" was a normal liver, the lower edge of which reached nearly to the umbilicus. When the patient was recumbent the organ assumed a less abnormal position. Both kidneys could be pressed out of the loins; the colon and small intestines appeared to be gathered together into a mass below the umbilicus. The stomach was much displaced downwards. The thorax was constricted about the lower part in the manner described on p. 589.

The patient denied tight-lacing at any period of life. The case was a pronounced example of visceroptosis; yet the patient had no abdominal symptoms of any kind. Her digestion was good and her bowels regular. She was a placid person who lived at her ease

and was at peace with herself and with all men. She stated that she was not strong, but that she lived carefully and enjoyed excellent health. The "tumour" had disturbed her in no way, and had merely aroused an amused curiosity. She was feebly pleased to hear that she could continue to take "carriage exercise."

2. An unmarried lady of thirty consulted me with regard to what she was assured was a stricture of the colon. She had the persistent pain and very marked tenderness at a spot just above and to the left of the umbilicus, to which I have just referred. The stricture was supposed to be situated at this spot. She had frequent vomiting which appeared in "attacks." The vomited matter was small in amount and usually intensely acid. Her bowels were confined, and she described her motions as containing many strange and wondrous things. An examination of one of these remarkable stools revealed nothing unusual except much undigested food and much mucus. The patient was thin and pallid. She was too weak to follow any other engrossing pursuit, but she had made a great study of her malady.

The abdominal pain had begun ten years previously, and since that time she had had manifold illnesses. She had written out an account of these disorders with much care. They seem all to have been of an acute and exceptional character, and included "malignant sore throat," "internal abscess," "spinal exhaustion," "neuralgia," and certain smaller troubles.

She had had uterine affections of bewildering complexity. She spoke of her kidneys with precision and of her liver with regret. The former organs were prone to unreasonable congestion, and the latter to an "obstinacy" which appears to have been little short of intelligent.

She had been to many health resorts and had taken "medicine" for the ten years concerned.

She was certainly feeble, anæmic and intensely neurotic. The abdominal pain was at times agonising; and her friends were driven to desperation by an illness which seemed unending and filled with tortures. The abdomen was flabby and revealed nothing beyond a general ptosis of the abdominal viscera of moderate degree. The kidneys were both slightly movable. As all medical measures had failed, as the patient was leading the miserable life of a chronic invalid, and as her friends were becoming convinced that "she *must* have something wrong," I consented to make an abdominal exploration. I found a general prolapse of the viscera of moderate but distinct degree; the stomach was perhaps a little dilated, but beyond this every organ in the abdomen was perfectly normal. The intestine was closely examined from the duodenum to the sigmoid flexure; it was perfectly normal.

The exploration cured the patient of all her abdominal symptoms, even of the vomiting and of the pain which had troubled her for ten years. I think it possible that the ptosis of the mass of intestines may have hindered the passage of food from the stomach in a trifling degree.

This case, therefore, was one of uncomplicated visceroptosis with nervous phenomena in a very neurotic patient. Although the patient had never worn a belt she was always worse when she moved about, and was only comfortable when in bed. A belt was worn after the operation.

3. A young lady, about twenty-two years old, had been ill for six years with distressing but somewhat indefinite abdominal troubles. There was a history of tuberculosis in the family. The patient was highly cultivated and intelligent, and had never exhibited any symptoms which could be considered "nervous," nor did she in any way conform to the conception of a neurotic subject.

Her troubles began six years ago with an acute abdominal disturbance, which had been ascribed to an ulcer of the stomach or small intestine.

She was never well after the illness. The symptoms were in a general way in accord with those associated with Glénard's disease. There were evidences of continued catarrh of the bowel. Abdominal pain was almost constant. Vomiting was frequent, and usually the vomited matter was intensely acid; it sometimes appeared to be composed solely of a very acid gastric juice. There were considerable digestive disturbances and still greater troubles with the bowels. The patient became much enfeebled.

It was soon evident that all the abdominal troubles were increased by movement and by the erect position. The patient was most comfortable —and, indeed, only comfortable—when lying down. In addition to dragging pains and a burning sensation about the stomach, there were the phenomena usually associated with movable kidney. The right kidney was found to be "floating," and was fixed by suturing by a well-known Continental surgeon. The operation was entirely successful.

The patient was continuously under treatment. Every form of diet was tried. Many spas and health resorts in various parts of Europe were visited, but with no other than temporary or doubtful benefit. It was considered at one time that the symptoms had a nervous origin, and the patient submitted to the Weir-Mitchell treatment by absolute isolation, massage, and special feeding. These measures, although persevered with for the full time agreed upon, rather aggravated the patient's condition, or were certainly attended by no improvement.

The young lady had for some time been compelled to wear a belt, and indeed, unless the belt were in position, she was unable to stand or move about without great discomfort. When first I saw her, the "belt" had been gradually increased in power and substance. It now consisted of a large shield-shaped metal plate to which two steel levers were attached, and by means of which the plate was made to bear pressure upon the lower part of the abdomen. This very formidable-looking apparatus had been gradually evolved by the patient to meet her need for increased support by pressure. When it was in position it altered the pulse in the lower limbs, and I condemned it as positively dangerous.

When examined in the recumbent position the area of hepatic dulness was normal; but when the examination was made in the erect posture the liver was found to have descended some two inches. The stomach and the whole mass of the intestines appeared also to fall downwards. There was a certain amount of resistance with pain and tenderness over the region of the transverse part of the duodenum. The spleen shared in the general ptosis. The left kidney could not be felt. When in the erect position much complaint was made of pressure upon the bladder.

I ventured to advise that an incision be made into the abdomen, partly to ascertain the condition of the viscera, and partly to fix the liver in position. This step did not seem to be premature, as the patient was in continual distress and was practically bedridden. Moreover, she seemed to have exhausted every measure of medical treatment, and her weakness and intestinal troubles were increasing. It was the opinion of those who had seen most of the patient during her long illness, that unless some speedy improvement were effected she could not live much longer.

I opened the abdomen in the median line a little below the xiphoid cartilage. The liver presented, and I found that I could drag it down to a quite remarkable extent. The stomach occupied a lower level than usual, but I found it impossible to drag it upwards so as to cause it to protrude at the wound. I introduced the hand, and found that the great omentum was rolled up into a round and rigid cord, and was fixed to a mass of stony hardness in the upper part of the right iliac region. I made a second incision directly over this mass, and found it made up of a collection of old tuberculous glands situated in the mesentery of the ileum. These glands were at once removed. Two of them were dry and caseous and presented calcareous foci; the third gland was wholly calcareous, and was indeed practically a stone. On returning to the median wound I found that the stomach could now be readily drawn up. I drew it through the wound for examination and could find nothing abnormal in it. The spleen was very mobile, and the transverse colon was entirely below the level of the umbilicus. About the descending part of the duodenum were some vague adhesions. An examination of the intestines, cæcum, and appendix, and of the pelvic organs revealed nothing abnormal. I secured the liver in place and was able, now that the stomach was free, to raise it above its normal level. I made use of three stout silk sutures. The most important stitch was passed through the liver near its edge and penetrated the round ligament, which afforded a most substantial holding. Above, the sutures were passed through the fibrous structures of the parietes by the side of the xiphoid cartilage.

The patient made an excellent recovery, which was without complication save that a little sutural abscess formed about one parietal stitch. The troubles depending upon the ptosis of the viscera vanished. There was no longer any pressure upon the bladder. The liver remained per-

fectly in place when the patient was erect. She could walk about without the old discomfort and without any artificial support.

It will be evident from these three illustrative cases—

1. That the viscera may be found in the condition known as abdominal ptosis and yet no symptoms appreciable by the patient be produced.

2. That a quite moderate degree of abdominal ptosis may be attended by a definite series of nervous symptoms, that these symptoms may be distressing and even alarming, and that they may be made to disappear by a measure of treatment (abdominal incision) which apparently can act through the nervous system only.

3. That the condition of abdominal ptosis may depend upon definite organic lesions within the abdomen, certain viscera being positively dragged out of place.

This relationship between subjective symptoms on the one hand and anatomical conditions on the other, is not unlike that met with in connection with movable kidney. In the case of a movable kidney the patients may be divided into three classes: (*a*) those who have a movable kidney and who are unconscious of it, and who remain in perfect health; (*b*) those in whom a neurotic tendency exists, and in whom the mobile viscus becomes the focus of a number of indefinite and bizarre nervous symptoms; (*c*) those who experience real trouble from the floating organ, and who have repeated attacks of pain due to torsion of the renal pedicle, or to stretching or bending of the same.

It is probable that among feeble and unhealthy women who lead inactive lives and who suffer from dyspepsia and constipation, this ptosis of the viscera is more common than is supposed. It is probable also that the condition, even if discovered, would, as a rule, call for little interference, if any. With many persons such a condition must be merely a part of their meagre individuality. In all civilised communities there is a pallid host of feeble folk who are flabby both in body and in mind, who can take little share in the business of the world, and who assume more or less pathetically the pose of chronic exhaustion.

What part the nervous system plays in the development of such symptoms it is beyond my province to inquire; nor is it necessary to discuss the malady as a "neurosis," nor to discover whether it be the cause or the product of "neurasthenia."

In some instances no treatment, beyond the use of a supporting belt, is demanded, as there are no subjective symptoms to treat. To wear a well-fitting belt is, to say the least of it, a wise precaution in the majority of cases in which enteroptosis is discovered.

The cases attended with nervous phenomena require, in addition to a reasonable amount of support, such treatment as is in vogue for the relief of the neurotic. An exploratory incision into the abdomen may in certain instances be a proper part of that treatment.

Until we know more about the conditions of pressure within the abdomen, and until we can explain why a mere exploratory incision will cure tuberculous peritonitis and relieve for a while the symptoms

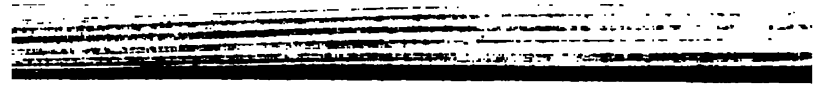
of undisturbed cancer of the pylorus, it will not be safe to assume that the operation acts merely by diverting the channels of a morbid imagination.

FREDERICK TREVES.

REFERENCES

1. GLÉNARD. *Lyon méd.* 1885; *Soc. méd. des hôp.* May 15, 1886.—2. GRASSET and RAUZIER. *Traité pratique des maladies du système nerveux*, 4th ed., vol. ii. p. 404. Paris, 1894.—3. RÜDINGER. *Topographisch-Chirurgische Anatomie*, 1878, plate viii. fig. A.

F. T.



DISEASES OF THE PERITONEUM

ACUTE PERITONITIS

Definition.—By the name "acute peritonitis" is implied an acute inflammation of the serous membrane which lines the abdominal cavity; and the name is considered to apply as well to cases in which the inflammation is general or diffuse, as to those in which it is more or less localised.

General considerations.—In dealing with the etiology of this affection, it will be pointed out that acute peritonitis is due, probably in all instances, to the influence of pathogenetic micro-organisms; it is a septic process. The invasion of the great serous cavity by noxious germs is immediately followed by the phenomena of inflammation, but those phenomena are not so all-engrossing as the name "peritonitis" leads us to infer.

Pain and tenderness, fever, and, so far as it can be shown, swelling are present. In cases of localised peritonitis these symptoms are certainly conspicuous; but when the trouble is more diffused they are very apt to be obliterated by the more conspicuous phenomena of general constitutional depression. In the most rapidly fatal cases the symptoms are distinctly those of poisoning rather than of inflammation. Almost always, indeed, there is about every patient who is dying of peritonitis the suggestion of a poisoned man. He lies back in bed prostrate, with gaunt cheeks and sunken eyes. There is a look of unceasing anxiousness in his face, and a sense of hopeless unquiet in his movements. His hands, which wander with pathetic restlessness over the bed-clothes, are cold and damp. The tongue is that of a man who is dying of thirst. There is constant vomiting. The breathing is laboured, and accompanied by faint sighs and groans, and the countenance is ashen and livid. So far as the aspect of the patient goes he might be dying from snake bite or from the poison of cholera, while there is little to suggest an inflammation so intense as to end fatally in a few distressful days.

When the patient dies, and the abdomen is opened, the amount of inflammation discovered is often out of all proportion to the phenomena which preceded death. In one case there may be merely some injection of the peritoneum, with loss of its polished surface, and in its place a general stickiness and a few frail and insignificant adhesions; in another there may be an ounce or so of thin greenish pus around the cæcum, an exudation, looking ill-conditioned enough to the surgical eye, but still a

mere scanty mixture of pus and turbid fluid, and not a formidable kind of effusion such as would be expected in a case of death from inflammation.

A further aspect of the case may be illustrated by certain experiments upon animals.

If a certain dose of a particular septic matter be introduced into the peritoneal cavity of an animal, peritonitis follows, of which the animal dies in three or four days, and there are found at the autopsy the characteristic changes in the serous membrane. If a smaller dose of the same poison be used, symptoms of peritonitis follow from which the animal may recover with or without the previous formation of an encysted intra-abdominal abscess. If a larger dose be employed, the animal may die within twenty-four hours with symptoms which are at least suggestive of peritonitis, but which are more evidently those of sepsis; after death the peritoneum is either devoid of any evidences of inflammation, or displays the most insignificant changes.

In like manner in the human subject some of the most rapid cases of death following such a lesion as is known to be capable of causing peritonitis may be found associated with but the very faintest evidence of inflammation within the abdomen. Such cases are undoubted examples of death from toxic infection, in which no time has been allowed for the establishment of inflammatory changes in the infected area.

As bearing further upon this matter, it is worthy of note that the cases in which the evidences of inflammation are most emphatic, and in which, indeed, suppuration is the most pronounced, are the very cases in which the prognosis is least unfavourable.

It would be in keeping with the phenomena met with in the majority of the cases of general peritonitis if that affection were defined as a septic intoxication depending upon infection of the peritoneum.

In connection with the manifestations of peritonitis certain facts concerning this great serous membrane have to be borne in mind.

(i.) Its surface is very extensive; it is probably, as Wegner states, as great as that represented by the whole integument of the body.

(ii.) The membrane possesses remarkable powers of absorption, as shown by the manner in which milk, blood, peptones, and the like, are taken up. It is said to be capable of absorbing an amount equal to from 3 to 8 per cent of the body weight. It is noticed that septic intoxication occurs with great rapidity when the peritoneum is concerned in the infection.

(iii.) The peritoneum offers some degree of resistance to septic organisms and their products. Experiments upon animals and clinical experience in man show that it can readily dispose of a certain quantity of septic germs. This important power varies within wide limits, and is at once compromised or abolished by injury to the delicate membrane. In experiments upon animals it is noted that pyogenetic cocci (for example the staphylococcus aureus) will act more readily and in smaller doses if introduced into the peritoneum together with some (sterile) corrosive fluid

capable of injuring the serous endothelium ; and further, that such injury to the peritoneal surface—in cases in which no cocci are injected—encourages the escape of the colon bacillus from the intestine.

(iv.) No tissue in the body is provided with more favourable conditions for healing than the peritoneum.

(v.) The peritoneum does not show the same degree of vulnerability in all parts, nor are all parts of it alike in their response to certain lesions.

The part which is apparently most sensitive to infection, and which is most prone to rapidly spreading and diffuse inflammation, is that which covers the small intestine.

The parietal peritoneum has certainly not so high a degree of susceptibility, and is not so apt to assume the diffuse and low form of inflammation. The morbid changes in peritonitis are more marked and more advanced on the visceral layers and omentum than upon the parietal part of the membrane, and this circumstance is not always to be accounted for by the point of origin of the trouble.

Terillon showed that the effect of irritation of the peritoneum is comparatively feeble over the parietal layer or over the liver, while it is violent and intense when the membrane covering the intestines is involved.

Localised forms of peritonitis and encysted exudations are comparatively uncommon in the large area occupied by the small intestine.

Localised peritonitis is met with in those regions which are more or less unoccupied by the coils of the lesser bowel, namely, in the subphrenic district between the dome of the diaphragm above and the transverse colon below, in the region of the cæcum, especially on the outer side of that region, and, lastly, in the pelvis. If a list be drawn up of cases of peritonitis in which recovery has either been spontaneous, or brought about by operation, in the great majority the disease will be seated in one or other of these three districts. It is certain that localised purulent collections are uncommon except in these situations.

The fatality of injuries of the intestine is well known, and the attendant peritonitis spreads with fearful rapidity.

Lesions of the liver and of the bile passages, on the other hand, are often followed by a peritonitis which is slow and moderate, and may end in recovery.

In making these comparisons it must not be forgotten that in the bowel lesion there will be greater opportunities for septic infection than in the case of a limited injury to the biliary passages, although in many instances of death from wound of the gut there has been no appreciable extravasation of its contents.

The movements of the small intestine tend to diffuse the infective material, but it is noteworthy that one of the first effects of peritonitis is to arrest all movement in the bowel.

(vi.) The general arrangement of the nerve-supply of the peritoneum is peculiar, and serves to explain certain phenomena in peritonitis.

The skin over the greater part of the abdomen, over that part at least beneath which lie the chief viscera, is supplied by the lower seven dorsal or intercostal nerves. The same nerves supply the muscles of the belly; namely, the rectus, the two oblique muscles, and the transversalis. Moreover they take a most important part in the nerve-supply of the peritoneum and of the abdominal viscera, inasmuch as the contribution which the great abdominal plexuses receive from the spinal nerves is derived in whole or in greater part from the lower seven dorsal nerves through the splanchnics. It is further to be observed that these particular spinal nerves are concerned in the movements of respiration, since they supply the lower series of intercostal muscles. Thus it happens that an arrangement exists for the most rapid possible conduction of reflex impulses. A cold hand is laid upon the abdomen, or a blow upon the part is threatened, and the muscles of the belly-wall become rigid in a moment. On the other hand, there may be some sudden lesion of the peritoneum or of one of the abdominal viscera, and at once the muscles of the belly-wall contract, the skin becomes more or less hyperæsthetic, and there is thus provided by the tender integument and by the rigid muscles that protection and that securing of rest which are the first elements in the natural treatment of a damaged part.

The normal peritoneum is possessed of great sensitiveness, and lesions of it are attended with very marked shock. When the surface of the serous membrane has become inflamed, and covered with lymph, the ordinary sensibility appears to be dulled or lost. The irritant which of all others appears to affect the peritoneum the most acutely is that represented by the invasion of the surface by noxious bacteria or their products.

(vii.) The last point concerns the possibility of the peritoneum acquiring some degree of immunity from septic infection.

It has been shown by Reichel and others that in dogs an immunity from peritoneal infection by pyogenetic cocci can be produced artificially. A small dose of septic material is introduced into the abdominal cavity, and after the animal has recovered from the disturbance a larger dose is injected; this process is repeated at intervals with increasing doses of the poison, until at last no symptoms are induced by a dose which in a control experiment would cause death within from twenty-four to thirty-six hours. An invulnerability not unlike this appears to be more or less possible in the human subject. Other things being equal, an operation carried out within the abdomen of a person who has had chronic peritonitis, or who has exhibited subacute attacks, and whose peritoneum presents substantial adhesions, is likely to be attended with better results than when the peritoneum is found wholly undisturbed.

Etiology.—*Varieties.*—It has now become evident that peritonitis depends almost entirely, if not entirely, upon infective processes, and that these agencies are concerned, directly or indirectly, in every form of the disease. The data upon which this statement is based are not yet

complete, but there is much presumptive evidence in favour of its accuracy, and the domain of non-infective peritonitis is becoming rapidly more and more restricted.

In dealing with any classification in which an infective process is the main element, there are many general considerations to be borne in mind. On the one hand there is the dose of the poison, and on the other hand there is the soil into which it is introduced. The dose must obviously be liable to considerable variations, not only in amount, but also in virulence; while the conditions offered for growth by the inoculated body must differ even to a wider and more complex extent. The resistance of the individual to the growth of septic organisms is influenced by a multitude of circumstances the value of which can scarcely be formulated. There are differences of sex and age and health, and even of race. There are such unfavourable conditions as Bright's disease, diabetes, and alcoholism. There are local conditions which lend themselves to the more ready cultivation of germs, such as extensive damage to the peritoneum and extensive interference with its power of dealing successfully with noxious micro-organisms. There are the presence of ascites and the coexistence of extravasations of blood, bile, or intestinal matter in the peritoneal cavity, foreign substances in the serous sac which may bring micro-organisms to the membrane, or may damage the delicate endothelium, and thereby neutralise the protective properties of the peritoneum, or at the least may form a convenient medium in which existing bacilli or cocci can grow.

The following is a classification of peritonitis according to its cause—

(*a*) Peritonitis due to infection from the intestine.

(*b*) Peritonitis due to infection from without.

(*c*) Peritonitis due to the pneumococcus.

(*d*) Tuberculous peritonitis.

(*e*) Peritonitis of doubtful origin, as, for example, those forms which are reported to be due directly to irritants, to rheumatism, or to gonorrhœa.

Any of the above-mentioned forms may be acute. The first three varieties are almost invariably acute. Tuberculous peritonitis, on the other hand, is more commonly chronic.

(*a*) *Peritonitis due to infection from the intestine.*—Under this heading are included most cases of peritonitis associated with hernia, with intestinal obstruction and with perforation; peritonitis due to any form of ulceration of the bowel; to enteritis; to cancerous growths of the gut, and to troubles in the vermiform appendix.

Peritonitis due to inflammatory changes in the biliary canals, and some forms of peritonitis following upon operation and injury, are also to be included.

The micro-organism which is usually found associated with these different forms of peritonitis is the bacillus coli communis. This remarkable bacillus was first described by Escherich in 1885. It has been shown to be identical with the bacillus pyogenes fœtidus of Passet, with the bacillus lactis ærogenes and with the pyogenetic urinary

bacterium described by Clado and Albarran in 1891. Though distinct from the bacillus of typhoid fever it closely resembles it. It exists normally in the human body and in healthy persons. It has been demonstrated along the whole of the alimentary canal from the mouth to the anus. It is indeed the most abundant and the most constant of the bacteria found normally in the human body. The bacillus is liable to considerable morphological variations or, as some suppose, many varieties are included under the one name. Like some other bacteria it varies greatly in virulence.

So far as experiments upon animals are of value, it would seem to be harmless when taken from the normal intestine. If, however, the bowel become the seat of certain diseased processes (or it may almost be said of any diseased process), then the bacillus becomes virulent at once, and if introduced into the peritoneum of an animal peritonitis follows.

Virulence has been found to be developed in cases in which the bowel was obstructed, strangulated, or inflamed, in venous congestion and œdema of the gut, in diarrhœa, in typhoid fever, in cholera, in the diarrhœa produced by purgatives, and in advanced constipation.

When the intestine is healthy the bacillus coli communis has little disposition to escape through the gut-wall or to invade the tissues of the body after death. If, however, the intestine be damaged or diseased, then the bacillus shows an active inclination to penetrate the wall, and is usually found to have extensively invaded the tissues after death.

This bacillus is pyogenetic, although its powers of inducing suppuration vary and appear often to remain dormant for a while. When peritonitis is induced by injecting a culture of the virulent bacillus into an animal it is usually of a purulent type. The colon bacillus in a state of almost pure culture has been found in man in the pus from an ischio-rectal abscess.

So far as the human subject is concerned, it has been shown that those various forms of peritonitis which are assumed to be of intestinal origin—including peritonitis starting from the biliary passages—depend mainly and in many instances solely upon the bacillus coli communis. This has been well shown in connection with peritonitis depending upon obstruction, or strangulation, or injury of the bowel; or due to perforation, to enteritis, to typhoid fever, to mischief in the appendix, or to cancerous growths in the gut. The colon bacillus may be found alone or in association with other micro-organisms such as streptococci or staphylococci, but in the latter instance it is very commonly the predominating microbe. In connection with this circumstance it has been suggested that the highly resistant colon bacillus may survive in an inflamed part after the primary organism which causes the trouble has died out, or has been crowded out by the invader (Welch). It has also to be borne in mind that pyogenetic cocci are rarely absent from the normal intestine; indeed in perforative peritonitis the streptococcus has often been found.

As regards peritonitis spreading from the biliary passages many authors

have shown that in the suppurative or ulcerative troubles of the gall-bladder or bile-ducts the colon bacillus is often the only bacterium found.

In the cases of peritonitis after laparotomy, in which the colon bacillus is found, it is probable that such an injury has been inflicted upon the intestinal peritoneum at the operation as to permit the microbe to escape through the bowel wall. We have seen that it requires but little damage to the bowel or to its serous covering to allow of such escape.

(*b*) *Peritonitis due to infection from without.*—This division includes puerperal peritonitis, peritonitis consequent upon inflammatory troubles in the genital organs or in the parietes of the abdomen, and some forms of peritonitis following upon operation.

The micro-organisms usually found in these forms of peritonitis are pyogenetic cocci and most commonly streptococci. The streptococcus may be found alone or be in association with other microbes, among which, however, it is the chief. It is the usual agent in puerperal peritonitis, and is thus active in the production of a very common and grave form of peritoneal inflammation.

The staphylococcus is rarely met with alone in cases of peritonitis, and is usually associated with the more virulent form of coccus.

Pyogenetic microbes of the kind now under consideration have been met with in peritonitis following operation, in that due to extension from inflammatory troubles in the parietes, and in peritonitis consequent upon pyosalpinx. As already stated, the streptococcus has been met with very conspicuously in perforative peritonitis, and also in many cases of perityphlitic abscess; in neither of these conditions, however, is it met with in pure culture.

Experiments upon animals have shown that injections of pyogenetic cocci into the abdominal cavity produce rapid peritonitis, and act more vigorously if the injection be mixed with a fluid difficult of absorption, or one capable of damaging the peritoneal endothelium.

In peritonitis following operation it is most probable that the streptococcus gains an entrance through the wound. In puerperal peritonitis, no doubt, it reaches the abdomen through the pelvic organs. In the rare examples of peritonitis associated with erysipelas of distant parts (such as the face) the streptococcus must be assumed to reach the serous membrane through the blood.

(*c*) *Peritonitis due to the pneumococcus.*—It has not been certainly shown that this variety of peritonitis exists.

In the first place, it must be noticed that peritonitis is a very rare sequel of pneumonia, although, on the other hand, pneumonia is a common complication of peritonitis.

In the second place, there is some confusion associated with the name "pneumococcus," and those who assert that this special form of peritonitis exists are not always precise in their indication of the micro-organism referred to. It would appear that the pneumococcus of Friedländer is a comparatively rare bacillus of little power; it has been found in broncho-pneumonia and in a few isolated cases of empyema. A

similar bacillus, the pneumococcus of Fraenkel (the micrococcus lanceolatus) is, on the other hand, of common occurrence and of considerable pathogenetic power. It has been found in the exudation in acute lobar pneumonia; it is pyogenetic, and has been found in association with acute abscess of the parotid, with otitis media, with arthritis, with empyema and other purulent conditions. It is often met with in the saliva and in the alimentary canal of healthy persons.

It is probable, therefore, that the micro-organism described in peritonitis is the micrococcus lanceolatus.

Many writers assert that they have met with cases of peritonitis in which the pneumococcus was without doubt the cause of the inflammation. The peritonitis in some of these cases was primary, and was the sole disease; in the other instances it was secondary to pneumonia. The pneumococcus would seem at one time to be noxious to the peritoneum, and at other times harmless. It is difficult to localise it in the peritoneum of animals by experiment. Cultures obtained from the exudation in certain instances of peritonitis in which the pneumococcus was the only micro-organism found, and injected into animals, caused death from general sepsis. Morisse details eight examples in which, and in the associated lesions, the pneumococcus was the only micro-organism found. In seven of these there was either pneumonia or pleurisy. The exudation was purulent in five, and sero-fibrinous in three.

(*d*) *Tuberculous peritonitis.*—This form of peritonitis depends upon the tubercle bacillus. It is more often chronic than acute, and is dealt with hereafter (p. 654).

(*e*) *Peritonitis of a doubtful nature.*—(*a*) Peritonitis due to irritants other than micro-organisms. The existence of a non-infective peritonitis is open to question, and is positively denied by many recent writers.

Peritonitis has followed the injection of sterilised croton oil or tincture of iodine into the abdominal cavities of animals. It has also followed the introduction of a piece of wadding soaked in 1 in 1000 solution of corrosive sublimate. All these substances tend to damage the peritoneum and to inflict such an injury to that membrane as will facilitate the escape of the colon bacillus from the intestine. Fraenkel's researches tend to show that in these experiments the peritonitis is thus induced, and is therefore really infective. A sterile irritant is introduced and peritonitis follows: if the effusion be examined at an early stage no micro-organisms are discovered; if, however, the case be advanced the exudation is purulent and swarming with bacteria which have passed through the bowel wall.

Fæces, if introduced into the peritoneal cavity, cause peritonitis not by chemical action, nor as an irritant, but by reason of the micro-organisms or their products contained in the extravasation. If the material be sterilised no effect follows. Fresh filtered fluid from the bowel when injected into the abdomen caused a fatal peritonitis in the exudation of which the colon bacillus was found. The same fluid when filtered through many layers of gauze set up abdominal symptoms, but all the animals

survived. When the fluid was sterilised it had no effect. The same results have attended the injection of human bile into the peritoneum of an animal. Normal bile is said to be sterile, but in inflammatory conditions of the bile passages the bacillus coli communis has been found. In man a considerable effusion of bile may take place into the peritoneal cavity without causing peritonitis.

It would appear that blood is not of itself capable of producing peritonitis, although it affords a very favourable nidus for the development of any bacteria which may gain access to the extravasation. If the peritoneal surface has been so damaged as to favour the escape of micro-organisms from the bowel then the presence of blood is a damaging complication.

(β) Peritonitis due to rheumatism.—Spillmann and Ganzinotty have collected fifteen recorded cases of rheumatic peritonitis with nine deaths. In some of these cases the peritonitis appeared alone, in others in association with rheumatic troubles in the joints, or in other serous membranes. The evidence, however, upon which these pathological assumptions depend is by no means satisfactory, and will not stand a critical examination. The matter is again discussed on pp. 633-4, and in the article on "Perityphlitis," p. 879.

(γ) Peritonitis due to the gonococcus.—Peritonitis has been met with in association with gonorrhœa, but inasmuch as the gonococcus cannot survive in the peritoneal cavity it is doubtful whether peritonitis due to the gonococcus alone has any existence. This micro-organism is pyogenetic, but in gonorrhœa it is commonly found that other cocci—such as the staphylococcus pyogenes aureus — are associated with it. It is very probable that some such mixed infection is the cause of the pyosalpinx often met with in gonorrhœa. That pyosalpinx may lead to acute peritonitis is undoubted, but it has not been demonstrated that such a complication is due to the action of the gonococcus alone; it is probably the outcome of a mixed infection such as that just alluded to. Interesting articles on this subject have been published by Horowitz, Menge, and Wertheim.

Other varieties of peritonitis are dealt with in the article on "Chronic Peritonitis," p. 638.

One Hundred Cases of Peritonitis from the Records of the London Hospital

Varieties of Peritonitis.		Number of Cases.	Sex.		Average Age.	Result.	
			Males.	Females.		Deaths.	Recoveries.
Due to infection from the intestine.	With hernia	10	3	7	52	10	...
	With perforation of bowel	9	4	5	40	9	...
	With gross disease of bowel	6	4	2	40	6	...
	With disease of appendix	22	19	3	22	6	16
Due to infection from without.	Starting from lesions about the pelvis	18	5	13	36	11	7
	Following abdominal wounds	14	2	12	36	14	...
	By extension from the pleura	1	...	1	32	1	...
Tuberculous.	Tuberculous	8	2	6	20	5	3
Of doubtful origin.		12	4	8	35	8	4
	TOTALS	100	43	57	...	70	30

Relative frequency, age, sex.—The above table, which deals with quite recent records, shows the distribution of 100 cases of peritonitis with regard to relative frequency, age, and sex. It will be seen that forty-seven cases are due to infection from the bowel, thirty-three to infection from without, while eight cases are classed as tuberculous, and twelve as of unknown origin. No example of peritonitis due to the pneumococcus comes in the list.

As regards *age*, it is to be observed that peritonitis is most common in adults between thirty and forty, that the oldest patients are represented by those who were the subjects of strangulated hernia, and the youngest by the subjects of tuberculous peritonitis. It is also to be noticed that peritonitis due to the appendix occurs in comparatively young subjects. Indeed the peritonitis of children and of young men and young women is usually due either to tubercle, to mischief in the appendix, or to acute mischief, such as intussusception or intestinal strangulation, within the abdomen.

Lesions starting in the pelvis are a common cause of peritonitis in women during the active period of child-bearing.

In the matter of *sex* peritonitis is more common in women than in men, the difference being mainly constituted by the greater frequency in women of pelvic troubles and of abdominal operations. Peritonitis due to mischief in the appendix is much more common in men than in

women; and if this variety of peritonitis be excluded, then it will be found that cases of peritoneal inflammation in women and men stand about in the proportion of two to one. Tuberculous peritonitis appears to be somewhat more common in the female than in the male sex.

Morbid anatomy.—The changes met with in peritonitis are, in all general points, identical with those met with in inflammation of other serous membranes, such as the pleura and the synovial membranes of joints.

The earliest changes are best seen by the surgeon in the course of operations for acute abdominal mischief. When the belly cavity is opened the intestines are found to be distended, and this especially applies to the lesser bowel. The distended coils look red and congested, the injection is apt to be in patches, and to be most distinct along the lines of contact between the opposed coils and loops; the serous surface is dull and sticky, and possibly a little thin, clear fluid may be noticed among the intestinal loops.

In these early stages the endothelial cells are found to be swollen, turbid and granular. By some writers they are described as undergoing multiplication. Beneath the disordered endothelium is an inflammatory effusion rich in leucocytes, and it is this effusion which soon obliterates the details of the serous surface.

As the trouble advances the exudation increases, and the peritoneum becomes covered with a very distinct layer of lymph. The thickness of this layer varies. It is most substantial along the lines of contact between the various coils of bowel. It tends to fill up these lines, to be prismatic on section, and to obliterate the gaps between the implicated loops. This lymph is at first clear, but soon becomes dull, opaque, and milky. Sometimes it is yellowish-gray, or even greenish. It is soft and is readily torn off in flakes of unequal thickness. It is to this condition that the name adhesive peritonitis is given.

The effusion of serum which accompanies the appearance of the lymph increases and may become considerable. The effused fluid is yellowish or greenish, and is usually rendered turbid by flocculi of detached lymph. Such is the condition of pressure within the abdomen that this effusion is found diffused among the intestines; it does not gravitate first to the pelvis as has been stated; the recumbent position, no doubt, does not encourage such gravitation. As met with during operation most of the fluid—if it be free—appears to be in the loin regions.

The exudation associated with the bacillus coli communis when it attacks the peritoneum is often characteristic. It is well seen in cases of inflammation about the appendix. The fluid is at first clear and greenish looking; it then becomes greenish-yellow, then semi-opaque and foul-smelling. If it pass on to suppuration the pus has no especial characters except that it is frequently offensive. In chronic cases there is sometimes produced a white, soft, custard-like material, which can be scraped away in considerable quantity from about the cæcum.

Speaking generally, the character of the effusion in peritonitis is liable

to very considerable variations depending upon the degree and stage of the trouble. In the cases which tend readily to recovery the fluid may remain scanty and clear. In the cases where progress is from bad to worse the fluid becomes opaque, and is described as sero-fibrinous or sero-purulent; it may remain thin and foul-smelling, even in advanced cases which end in death. There may be a production of actual pus, which may be mixed with a thin acrid fluid, or may be thick and laudable, or be peculiarly offensive. The terms applied to the exudation in peritonitis show by their great variety how manifold are the conditions which the effusion may exhibit. If perforation has taken place the effusion may present some of the contents of the stomach, the intestine, the bladder, or the biliary passages. There may be an admixture of blood with the exudation, and hæmorrhages beneath the serous membrane are not uncommon. There is nearly always a certain amount of subperitoneal œdema. The bowel wall is infiltrated. The capsule of such organs as the spleen is thickened and opaque; the fluid produced in the course of the inflammation may be diffused generally throughout the abdomen, or be limited by adhesion to a particular district. In this so-called "encysted peritonitis" a considerable quantity of pus may be pent up in the iliac fossa within the pelvis, or in the sub-diaphragmatic region.

In many of the fatal cases the patient dies before any pus is produced, or indeed while the effusion is still clear. Actual pus is not common in the cases of non-localised peritonitis. In many of the examples of "sero-purulent" effusion no pus cells are to be found.

The fluid which is encysted need not be purulent; a clear effusion may be found so localised.

In case of recovery the restitution of the parts to their normal condition may be quite complete. The fluid is absorbed, the exuded lymph undergoes fatty degeneration and vanishes totally; on the other hand this lymph exudation may be replaced by granulation tissue which may become organised and produce definite "adhesions." The extent of these adhesions is liable to infinite variation; at first they are membranous, then as peristaltic movements pass constantly along the intestines these membranous adhesions become attenuated and stretched, and many give way entirely, and persist only as minute "tags" upon the bowel. Others are drawn out to some length, and are moulded by the ceaseless rolling of the intestine into roundish cords. Such cords or bands may be the cause of intestinal obstruction. In other instances the adhesions are short, wide, and extensive; parts are matted together closely, or the features of the various organs concerned may be lost in a confused conglomeration.

Symptoms.—The clinical phenomena of acute peritonitis are liable to considerable variation, not only in the matter of intensity and in the relative prominence of individual symptoms, but also in the fluctuations which those symptoms may show.

In some examples—and among these are the most acute cases—the

phenomena are, in the main, those of septic poisoning. In other instances the local symptoms attract most attention and take a predominant place.

It will be convenient to consider, first, an ordinary case of acute peritonitis; secondly, the several symptoms; and, thirdly, the clinical varieties of peritonitis.

I. *An ordinary case of acute peritonitis.*—The onset is more or less abrupt, and there may be an initial rigor. The first symptom is abdominal pain, which becomes rapidly diffused, and is of an intense and even agonising character. More or less prostration and shock are present. The abdominal wall, which at first may be retracted, becomes rigid and board-like, and the skin surface is markedly hyperæsthetic. The respiration becomes costal. Nausea and vomiting appear early. The vomited matter is small in amount, is frequently ejected, and does not commonly become fæculent, although it may be very offensive. The bowels are confined, and the constipation is as a rule absolute. Meteorism appears at an early period, and the rigid abdomen becomes tense and tympanitic on percussion. The patient lies with the knees drawn up and with the hands above the head. There is much restlessness and much thirst. Some degree of fever is usual. The pulse is rapid, small, hard, and wiry. The tongue is coated and foul, and later becomes very dry. Prostration becomes pronounced. The face is pinched and the eyes are sunken. The whole aspect is that of painful exhaustion.

If the case end fatally, the pulse gradually fails, becoming at the same time more and more rapid, and at last uncountable. The prostration is extreme. The face becomes more and more ashen, and often cyanotic. The limbs become cold and blue, and, together with the face, are covered with a chilly perspiration. When the surface is already quite cold a thermometer in the rectum may register several degrees above normal. The respirations are more hurried and shallower, and the voice is weak or lost. Even when the pulse can no longer be felt, the patient, now ghastly with his sunken eyes and hollow cheeks, retains his piteous restlessness. He gradually sinks, and as a rule retains his consciousness almost to the last.

Many of the early symptoms may disappear some time before death. The pain may vanish, the tenderness may entirely leave the abdomen, the bowels may act, and the patient may consider himself very much better. The vomiting may cease to be urgent, but it very rarely disappears altogether, and very often in the act of dying there is a great gush of foul brownish fluid from the mouth and nose of the now probably unconscious patient. The meteorism is another symptom which seldom shows amelioration. However distressing the aspect of the patient may appear, there are reasons to believe that death from acute peritonitis is not so full of suffering as might be supposed.

II. *The several symptoms.—The Onset.*—The onset of the symptoms in acute peritonitis is usually abrupt. This is especially so in instances of perforation in individuals who, at the time, are not acutely ill. In peritonitis due to lesion of the appendix the appearance of the inflam-

matory phenomena is commonly sudden. In puerperal peritonitis, in peritonitis due to operation, or to extension from adjacent parts, the onset may be not actually abrupt, but there is usually little difficulty in deciding the moment when peritonitis sets in. When the inflammation of the serous membrane follows typhoid fever, strangulated hernia, or intestinal obstruction, the precise beginning of the peritonitis may be masked by the previous symptoms, and one series of grave symptoms may merge into the other. In cases of acute intestinal obstruction, for example, it is often difficult to indicate the exact time when peritonitis began to complicate a condition already serious and disastrous enough.

A rigor does not often mark the onset of peritonitis. Among the 100 cases dealt with in table (p. 610) this symptom was recorded in thirteen instances only. In two of these the peritonitis had started in the appendix, in one it followed laparotomy, and in the remaining ten it had its origin in the pelvis. In may be mentioned that out of the thirteen patients ten died. Repeated and violent rigors are exceedingly uncommon.

The pain and the condition of the abdominal wall.—The pain in peritonitis is probably the earliest symptom. It is usually exceedingly severe, and is described as agonising, as "burning" or "cutting," or as a "tearing at the bowels." Some patients describe it as a terrible sense of fulness, as if the bowels were distended to bursting; they often have the impression that if flatus could be passed they would be much relieved. The pain does not bend the patient double as colic does, nor is it relieved by pressure; often, however, it is marked by exacerbations, especially in the early stages.

This pain as a rule is not localised at first. It is complained of as "all over the belly," or is referred to the region about, or the part above, the umbilicus. In fact the patient refers the pain to the great abdominal nerve-centres, and will sometimes place his hand or finger directly over the site of the solar or superior mesenteric plexus. As the symptoms progress the localisation of the pain becomes usually more and more precise, assuming that the trouble remains to a certain extent local. Thus in perityphlitis the patient will at the onset indicate the seat of the pain as vaguely about the umbilicus. Later he will very often point to it as immediately in the region of the disordered appendix.

As the pain increases in intensity the patient often speaks of it as "spreading."

The contraction of the abdominal muscles is well marked in nearly every instance; the belly becomes board-like. In certain examples of perforative peritonitis the contraction of the belly wall may be so extreme that the parietes sink in and appear almost as if resting upon the spine. This great retraction of the belly is comparable with that sometimes met with in lead colic. It has been for the most part noticed in connection with perforation of the stomach. In the less acute or less sudden cases this contraction is not so marked. It always tends to relax as the case advances. The rigidity of the abdominal muscles fixes the

abdomen, and gives a firm, protecting cover to the inflamed part. It tends also to give rest to the disturbed region by preventing movement. Not only is the belly made rigid, but the whole of the trunk of the body becomes to some degree immovable, and although the patient may move his limbs about, he keeps the trunk stiff.

As a result of this muscular contraction, the abdominal respiratory movements are greatly restricted, and the lower ribs are prevented from taking part in the act of breathing. By this means, again, a further element of rest is bestowed upon the parts within the abdomen. It will be observed that in inspiration the lower intercostal spaces do not fall in. If the contraction of the muscles be marked and the patient thin, the outline of the rectus muscle may become quite evident.

Another symptom usually contemporary with the onset of the pain is great tenderness of the abdomen. This is often extreme, and consists in an actual hyperæsthesia of the surface, rather than in tenderness within the abdomen. In some cases the hyperæsthesia is so extreme that the patient cannot bear the touch of the lightest hand, and is even afraid to allow the bed-clothes to come in contact with the skin of the abdomen. In such instances it will now and then be found that, although a light touch is acutely resented, deep pressure can be borne with comparatively little complaint.

The symptoms now dealt with, namely, pain, rigidity of the abdomen, and hyperæsthesia of the surface, are all intimately associated. They depend really upon one nerve disturbance. As already pointed out, the same nerves which go to the peritoneum supply also the muscles of the abdominal wall, the lower intercostal muscles, and the skin covering the front of the abdomen. Other things being equal, the more severe the impression made upon the visceral nerves, the more marked will be the contraction of the muscles and the hyperæsthesia of the skin. These symptoms all point to rest for the inflamed part; and the tenderness of the surface has this effect, that the patient will not tolerate any pressure upon the abdomen, or any movement—such as rubbing—applied from outside. This is a good thing, because a rough surgical examination may serve to reopen a minute perforation which had just been sealed by a flake of the omentum.

The first demand of nature then in acute peritonitis is a demand for absolute rest.

The pain, the rigidity of the belly, and the sensitiveness of the surface vary within wide limits, and are most marked in cases which are sudden and acute, and especially in those in which the peritoneum at the time of the outbreak was perfectly normal. The nerve condition of the individual has also to be taken into account. In chronic cases, in cases complicated with ascites, and in cases in which a sudden trouble falls upon a peritoneum which has long been exposed to irritation, the phenomena may be little marked.

As the peritonitis advances, both the hyperæsthesia of the skin and the contraction of the belly wall lessen, and may pass away almost

entirely. The pain also has a tendency to decrease. As the muscles yield the phenomena of meteorism usually appear, and any tenseness of the abdomen which remains depends upon flatulent distension of the intestines. If general septic symptoms become marked, both the contraction and the tenderness usually disappear. I have seen cases of septic peritonitis run their entire course with a flaccid belly wall, and with scarcely any tenderness. In such instances the patient is usually past middle life. I have known such a person spend some hours in applying massage to his own abdomen.

Shock.—Certain symptoms, which are collectively known as the phenomena of shock, mark, almost without exception, the clinical beginnings of those cases of peritonitis which are abrupt in their onset, or acute, or even subacute in their course. These symptoms are evidently due to an impression upon the nervous system, and are independent of inflammation on the one hand or of septic intoxication on the other. The shock attending a severe peritoneal lesion may prove fatal, and the patient may die with evidence of no important disturbance other than that wrought directly through the great nerve-centres.

The signs of sudden and grave disturbance of the peritoneum are pain, profound exhaustion, a distressful anxiety, pallor, a small soft quick pulse, cold extremities, shallow respiration, and vomiting. The phenomena vary in degree, and are not absolutely invariable in character. They often mark the earliest symptoms of an acute and suddenly produced peritonitis, or rather indicate the occurrence of a lesion which will lead to peritonitis. It is important to note that, in some degree, these symptoms are common to all cases in which there has been a wide and abrupt impression made upon the nerve-centres within the abdomen. It may almost be said that all quite acute troubles within the abdomen begin with the same train of symptoms. Until some hours have elapsed, it is often impossible to say whether a sudden abdominal crisis is due to the perforation of a vermiform appendix, to the bursting of a pyosalpinx, to the strangulation of a loop of intestine, or to the passage of a gall-stone. The twisting of the pedicle of an ovarian cyst has led to symptoms which have been mistaken for peritonitis; a sudden peritoneal hæmorrhage has been confused with intestinal obstruction, and the rupture of a hydatid cyst has been diagnosed as a perforation of the intestine. It is quite possible, quite usual indeed, for these various troubles to present, at first, symptoms which are common to them all, and which merely indicate that a shock has been communicated to the great abdominal nervous system. To these common phenomena of a crisis within the abdomen Gübler has applied the convenient name of "peritonism." [*Vide* art. "Shock and Collapse," p. 320.]

Vomiting is one of the most frequent and most marked of the symptoms of peritonitis; it is generally one of the most distressing features of the trouble. Out of the hundred hospital cases (see p. 610), in only twenty-three is the vomiting spoken of as slight. Vomiting

appears early in peritonitis, and is indeed one of the very earliest symptoms. It may be preceded by nausea. The matter vomited consists at first of the contents of the stomach, then it becomes bile-stained, bitter, and often of a spinach green colour. Later still it becomes brownish, and has that horribly sickly smell which is generally called "intestinal." Finally the matter ejected may have a distinctly fæculent odour.

The vomiting is frequent, and the matter brought up is small in amount, the patient very commonly bringing up only a mouthful at a time. The act of vomiting is often spasmodic, is attended with little effort, and is followed by no relief. In only quite rare instances can the vomited matter be spoken of as copious.

Between the attacks of vomiting there may be eructations of foul gas.

So long as vomiting continues the patient is not improving.

In some of the more insidious septic cases, and in examples of peritonitis in the aged or in the subjects of advanced visceral disease, the vomiting often amounts to little more than a slight irritability of the stomach.

In the instances in which the sickness is excessive, and in which the vomited matter becomes "intestinal," something more than a mere reflex disturbance is at work.

In the cases of peritonitis following strangulated hernia the vomiting is for the most part very marked and often copious, but it bears no constant relation either to the state of the gut, the degree of peritonitis, or the height of the fever.

In perforative peritonitis all the symptoms are usually acute and pronounced. There is intense pain and a corresponding degree of collapse, and in the larger proportion of the cases vomiting is conspicuous; but it is usually absent in examples of perforation of the stomach. In the cases connected with typhoid fever the relation between the vomiting and the action of the bowels is notable. If all action of the bowels ceases, the vomiting then tends to be marked; but if the loose motions continue, then the sickness is for the most part slight. In peritonitis depending upon disease of the intestine, including mischief in the appendix, vomiting, as a rule, is not a pronounced symptom. In the cases of perityphlitis it is to be noted that it is usually least marked in the cases in which the bowels act with more or less regularity.

In peritonitis taking its origin from the pelvic viscera or parietes, vomiting is seldom very distressing.

In the acute cases the symptoms are rather those of septicæmia, and in the chronic cases the disturbance has a great disposition to remain localised.

In peritonitis following operation or accidental wounds, vomiting is rarely absent; it may not be marked, and may consist merely of an irritability of the stomach.

Like this symptom in other cases of peritonitis the gravity of the vomiting does not depend either upon its frequency or the amount ejected, but upon its deadly persistence.

Meteorism is not among the very earliest phenomena of peritonitis, although its appearance may not be long delayed. It sets in, as a rule, within twenty-four or forty-eight hours. Its degree varies considerably, and is influenced by conditions which are not yet fully understood. When it exists its appearance is coincident with that relaxation of the contracted abdominal muscles to which allusion has been already made (p. 615). Meteorism may be absent throughout the whole progress of the case, and examples of this kind are not uncommon in cases of septic intoxication attended with symptoms of a low type. It is most marked in peritonitis attended by actual intestinal obstruction, in examples of perforation, in cases in which there is thrombosis of the mesenteric vessels, and also in cases in which opium has been freely administered.

The meteorism is first noticed, as a rule, just above the umbilicus, where it begins as a vague transverse swelling. In these stages it is often said that "the transverse colon is distended," although the tympanitic condition probably concerns the upper part of the small intestine. In due course the whole abdomen becomes evenly swollen and drum-like. Meteorism appears to present no definable relation to the amount or degree of the vomiting; it is a condition coincident with absolute paralysis of the bowel. One of the first effects of peritonitis is to check all peristaltic movement; this arrest may not be absolute at first, but it is probably quite complete when the stage of meteorism is reached. In experiments in which artificial distension of the gut has been brought about, not only do all intestinal movements cease, but peristalsis cannot be induced by any of the usual irritants.

It is needless to say that this meteorism does not depend upon any mechanical obstruction of the bowel. It is probably caused in peritonitis by changes induced in the intestinal blood circulation. In any cases of peritonitis with symptoms otherwise mild and hopeful, persistence of meteorism is always an evil feature; this meteorism will persist even when the bowels act regularly.

Constipation.—Cessation of the action of the bowels is a conspicuous feature in peritonitis. In the majority of instances the constipation is complete. Flatus may be passed throughout the progress of the malady, and fæcal matter may now and then be washed away by an enema. A motion is often passed at the very beginning of the trouble, and before the symptoms have become very acute.

In 100 cases of peritonitis from the London Hospital (see p. 610) the bowels are classed as "loose" in twenty-eight instances. In some of these twenty-eight cases there was actual diarrhœa, in others the bowels acted with frequency and without artificial aid, while in a third series of cases a free action was maintained by enemas or by aperients. A loose action of the bowels appears to be most often met with in connection with pelvic peritonitis, which name will include puerperal peritonitis. Next in frequency it is found associated with peritonitis having its origin in the intestine, especially in connection with typhoid fever. It is not uncommon in the earliest stages of peritonitis—when

the symptoms are not very acute—for the patient to experience a repeated desire to empty the bowel. This is usually unavailing however.

It has not been made clear that the prognosis is definitely affected by the presence or absence of constipation (see, however, art. "Perityphlitis" in this volume).

Fever.—The temperature in peritonitis is liable to fluctuations so numerous and extreme that it is difficult, if not impossible, to construct any pattern of temperature which may be considered as characteristic of the disease. Probably there is no affection attended with fever in which the temperature charts of a large series of cases exhibit fewer data for the establishment of a characteristic course of fever.

A careful examination and analysis of the temperatures recorded in 100 cases from the London Hospital makes it evident that the one symptom of fever can afford very little guide in estimating the gravity of the case or in forming a dogmatic prognosis. A comparison of the temperature charts in the cases that end in recovery with those that end in death leads only to bewildering results. In some of the fatal cases the temperature moved steadily upwards, in others steadily downwards; in a third series of cases an even line of high fever was followed, while in a fourth set the temperature was about normal or lower. It is quite evident that no marked or regular relation exists between the range of temperature and the character of the peritoneal effusion.

In very general terms it may be said that perforation leads at first to a sudden drop in temperature, and that if the body heat be at the time high the onset of diarrhœa is associated with a more or less rapid diminution of fever. Moreover, it may be remarked that the chart record which carries with it the stamp of an almost inevitably fatal result is that in which the temperature is below normal for consecutive days.

The evidences of the most active fever are commonly seen in cases in which a plastic peritonitis is present, or one leading to well-encapsuled pus; while the examples associated with a low range of temperature often mark the cases in which a general sepsis (as from the intestine) is more pronounced than the peritoneal inflammation.

The details of the temperature can best be illustrated by reference to the various forms of peritonitis.

In peritonitis due to hernia the temperature is for the most part low, and in the majority of the cases does not rise above 99·5°. Chart No. 1 (from the case of a woman, aged fifty-five, in whom a femoral hernia had been strangulated for three days before the operation) is fairly typical. In the autopsy the bowel was found to be slightly ulcerated. The other viscera were sound. Before death the temperature in these—as in other marked septic cases—usually sinks lower and lower. Occasionally just before death it may spring up to 105°, as shown in Chart No. 2 (from a case of ventral hernia strangulated two days in a woman of thirty-five; the gut was black, but not perforated). This sudden elevation of temperature has been ascribed by some to a disturbance of the heat-con-

trolling centres by the circulation through them of blood charged with septic matter.

In the hernia cases the temperature ran highest (99° to 102° or 103°) in those instances in which the gut reduced into the abdomen was virtually gangrenous, or was extensively ulcerated. The onset of diarrhœa leads to a fall in the temperature.

In peritonitis due to perforation the temperature in the most rapid cases is that of collapse. If the temperature at the time of the perforation be high, it may slowly sink, especially in cases in which the bowels continue to act freely; or it may sink for a time and then rise again, as is commonly noticed in examples in which all action of the bowels ceases after the perforation. This is shown in Chart No. 3, from a lad of nineteen, with typhoid fever, in whom perforation took place on the eighteenth day.

When the perforation is slowly brought about, the temperature may rise at first, and then sink slowly towards death.

Chart No. 4 shows the four-hour record of fever in the case of a lad of eighteen, who was stabbed in the abdomen, and in whom—after death—a small puncture in the ascending colon was discovered.

In peritonitis attended with gross disease of the intestine the temperature is disposed to rise steadily, and then to drop somewhat abruptly before death. With actual gangrene of the bowel a high range of fever is common, and with profuse diarrhœa a lower range.

Chart No. 5 belongs to the case of a woman, aged forty-nine, with a growth in the descending colon, and ulceration of the bowel above it (there was no perforation). Peritonitis set in where the chart begins. On the third day a copious diarrhœa began, and its apparent effect upon the temperature shows a common condition.

In peritonitis due to mischief in the appendix the fever is usually high and of the ordinary inflammatory type. Its average range is from 99° to 102° or 103°. Its duration varies greatly, and a lower range of temperature is generally met with in the cases attended by diarrhœa.

Chart No. 6 is from a case of perityphlitis in a man, aged forty-four, in whom the earliest symptom was diarrhœa, and in whom the bowels were kept open throughout by enemas.

Chart No. 7 is from a case of perityphlitis in a youth of seventeen, who was treated with opium, and in whom there was very marked constipation.

In peritonitis, starting from the pelvis (with which is included puerperal peritonitis), the temperature is rather that of septicæmia. Chart No. 8 has been selected as fairly typical. It belongs to the case of a man, aged fifty-three, who was attacked by peritonitis on the seventh day, after a simple operation for fistula in ano. The trouble began with a rigor. The autopsy revealed a general sero-purulent peritonitis with septic thrombosis of the pelvic veins.

In examples of peritonitis following wound of the abdomen, the temperature is for the most part fairly high, running on an average

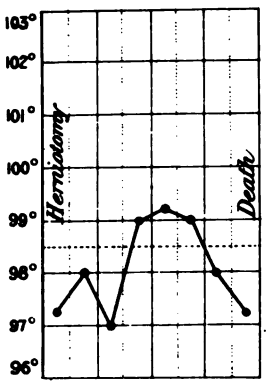


Chart No. 1.

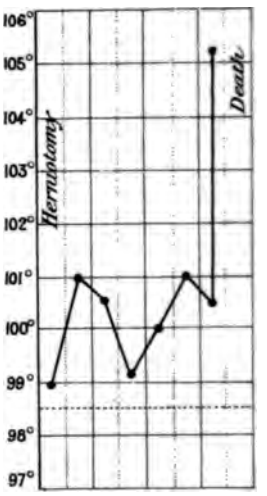


Chart No. 2.

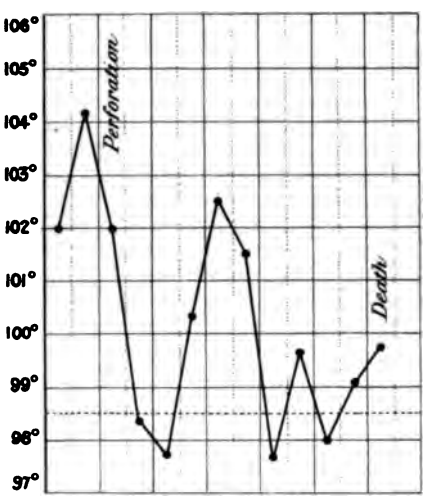


Chart No. 3.

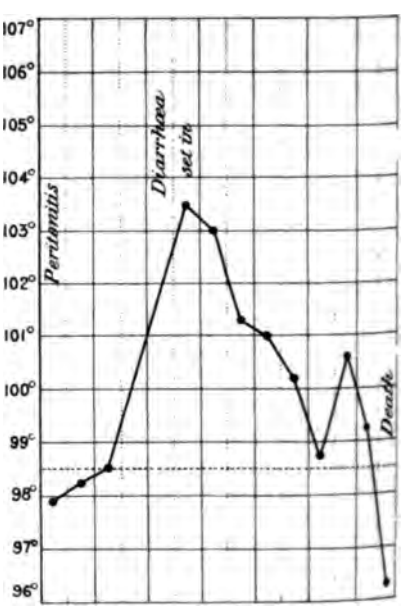


Chart No. 5.

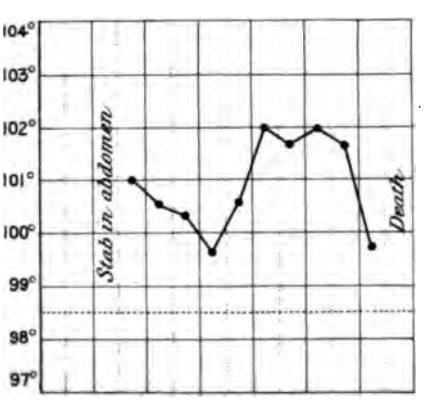


Chart No. 4.

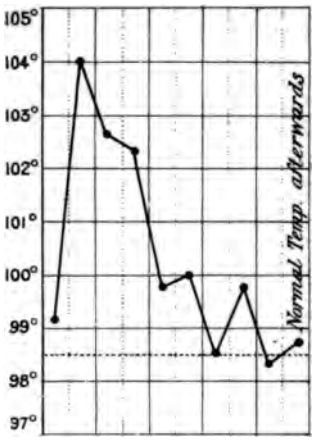


Chart No. 6.

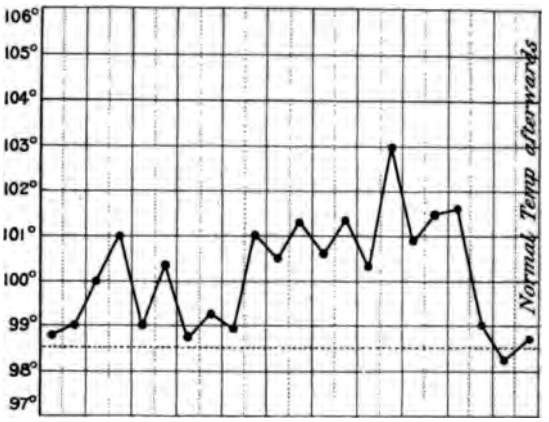


Chart No 7.

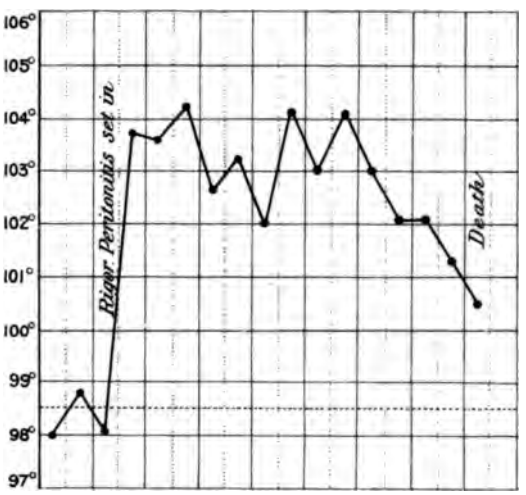


Chart No 8.

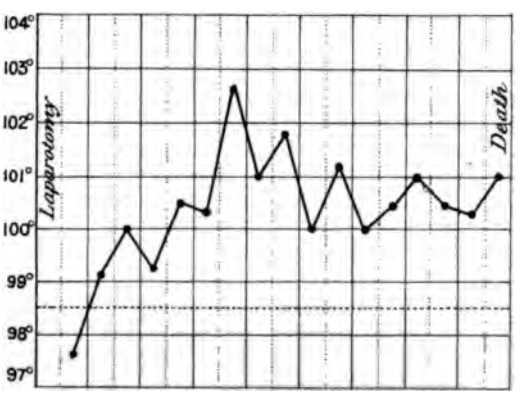


Chart No 9.

To follow

between 99° and 101° to 102°. In two cases from the London Hospital series, it remained below normal throughout. Chart No. 9 is selected as an example of an uncomplicated case following laparotomy. The patient was a woman aged twenty-six. The operation consisted in suturing a hydatid cyst to the abdominal wall. A general plastic peritonitis followed without any visceral complications.

General condition of the patient.—In the early stages of acute peritonitis the general condition of the patient is one of prostration due to pain and the torment of vomiting; later, in the event of a gradual improvement, these symptoms may give way, or, if the case end badly, they will pass into those which mark septic poisoning. In nearly all of the graver cases the phenomena of such poisoning predominate, and—as already pointed out at the beginning of this article—the patient presents the aspect of a poisoned man.

The attitude of the body in peritonitis soon becomes characteristic. The patient lies upon the back keeping the abdomen immovable, while the knees are drawn up and the hands—when not tossing about—are kept above the head. The flexing of the thighs helps to take some tension off the abdominal wall, and the carrying of the hands above the head tends to increase the capacity of the thorax.

The respirations are hurried—sometimes even 40 to 60—are shallow, and are almost purely costal. In women especially abdominal respiration very nearly ceases.

Hiccough is not uncommon. It is a very distressing symptom, tends to appear late, and often marks a serious feature in the prognosis.

The skin is dry and hot at first, but later it becomes cold and clammy.

The patient's face is pale and pinched (*face grippée*), the naso-labial crease is deepened, the forehead is wrinkled, the expression is one of pain, weariness, and anxiety. The eyes appear sunken and staring, the cheeks become hollow, the mouth is usually kept open. The tongue soon becomes white and coated, and then very foul, and later is dry and red or brown. It may be cracked. Sordes may appear upon the teeth in the later stages. Much complaint is made of the horrible taste in the mouth and of its dryness. The appetite is lost, but in its place there is a terrible and burning thirst, a symptom which is often most distressing.

The breath is peculiarly offensive. The pulse becomes small, sharp, wiry, or thready, and will vary at first from 100 to 120, and later will run between 120 and 160. The urine is scanty, concentrated, and high-coloured, and usually shows a copious deposit of urates. Micturition may be frequent, or there may be retention; but both these symptoms are uncommon.

Headache is often complained of. Sleeplessness is a distressing feature, and is associated with the restlessness which is so common in peritonitis. Delirium is uncommon even in the later stages. In the majority of instances the patient's intellect remains clear up to the last. Jaundice of slight degree is occasionally observed.

The examination of the abdomen.—From the account already given, the state of the abdomen, as revealed by examination, may be surmised. As meteorism increases, the abdomen becomes markedly tympanitic. The tympanitic note is more or less general, although it varies a little in degree in different parts. The areas of hepatic and splenic dulness are curtailed, and the latter may be annulled. If fluid effusion exists, dulness becomes—when the peritonitis is general—evident first in the loins. The loin will be dull on percussion up to a certain level; if the patient be turned over to the opposite side the part becomes tympanitic; if he be turned upon the side under examination the area of dulness becomes increased. In the sitting posture the whole of the dulness may vanish. In localised cases the effusion is limited to a certain area, as in perityphlitis; and its extent can be defined more or less readily by percussion. Auscultation of the abdomen reveals no sign of any value. It will be observed that all movements of the intestines have ceased, that the respirations are thoracic, and that in excessive meteorism the lungs are compressed and the heart displaced upwards.

III. *Clinical varieties of peritonitis.*—As may be imagined the clinical manifestations of acute peritonitis are liable to great variation. This must be so if it be remembered that the name "acute peritonitis" will include, on the one hand, a limited trouble due to torsion of the vermiform appendix, and on the other such an extensive lesion as will follow a large perforation of the stomach.

Much also depends upon the abruptness and violence of the onset. In some examples the patient is apparently in sound health at the moment that the peritonitis breaks forth, while in other instances the serous inflammation follows upon the later stages of intestinal obstruction, or upon such an exhausting disease as typhoid fever. In certain of the cases in the latter category the peritonitis is rendered almost unimportant in the face of the advancing disease and the evidently desperate position of the patient. In many instances of acute intestinal obstruction it is hardly possible to say when the peritonitis actually sets in, nor is its onset a matter of very great importance.

In general terms it may be said that most of the cases of acute peritonitis may be clinically arranged into three classes: i. There are cases in which the onset is very violent, in which shock is profound, and in which the patient dies in twenty-four or thirty-six hours with symptoms of utter collapse. Such a variety may be illustrated by peritonitis depending upon the rupture of a large ulcer of the stomach. ii. There are cases in which the manifestations throughout are those of septic poisoning, and in which the patient succumbs not so much to shock or to inflammation as to actual blood-poisoning. This is illustrated by some examples of peritonitis following upon strangulated hernia, or by puerperal peritonitis. iii. There are cases in which the trouble is localised, in which the local manifestations become prominent and ultimately predominant, and in which a definite effusion—which commonly becomes purulent—can be made out. An example of such a form is provided

by an acute perityphlitic abscess depending upon trouble in the appendix.

Peritonitis in children.—Acute peritonitis is always extremely grave in infants and young children. Constipation is somewhat less common than in adults. Vomiting is a less conspicuous feature. Meteorism is usually marked, and fever is high. The child utters a short cry or whine. No matter how severe the pain there is no loud or long-continued crying. Convulsions are not uncommon.

Masked peritonitis. — Peritonitis may run its course and end in death, and yet the symptoms be but very faintly marked, or be equivocal. This is not uncommon in the old and broken-down. Such patients soon sink into an apathetic, "typhoid" condition, complaining of little or no pain, and scarcely troubled with vomiting, while motions may be passed unconsciously in bed. I have seen such patients pat the abdomen to show that there is no tenderness in the region. Meteorism in such cases is seldom absent, and the rapidity with which the patient breaks up is often remarkable. The tongue soon becomes dry, the pulse is uncountable, the extremities become cold, and signs of death become rapidly evident. In the subjects of Bright's disease, and in characteristic patients who are saturated with morphia, a like obliteration of symptoms may be noted; the same also may be seen in peritonitis concurring with pyæmia. Now and then I have seen the same masked form of peritonitis follow upon operation in patients who were under middle-age, and who were constitutionally healthy. In two of these instances little or no pain was complained of, and little morphia was called for; the vomiting was quite insignificant, tenderness of the belly was absent, and meteorism was scarcely perceptible. The one predominating symptom was a never-ceasing and extravagant restlessness which appeared to wear the patient out, and which ceased only with death about the sixth day.

In a third case there was slight fever and very marked meteorism, but no pain and no vomiting. The tongue kept suspiciously foul. Food was well taken and the bowels acted well. Unceasing restlessness was the most marked feature. The pulse failed, increasing in rapidity on the sixth day; death took place on the seventh day.

It may be safely stated that in any form of acute peritonitis extreme restlessness is a symptom of very grave import. In those very acute cases in which the symptoms of poisoning are predominant the classical signs of peritonitis may be almost lacking.

Special forms of peritonitis. — *Local peritonitis* is well illustrated by perityphlitis (p. 890). *Puerperal peritonitis* and *pelvic peritonitis* are not dealt with in this work. [Vide *System of Gynæcology*, p. 485.]

The leading phenomena of *perforative peritonitis* are discussed in the sections concerning perforation of the stomach, and of the intestine. References may also be made to the articles "Intestinal Obstruction" and "Enteric Fever."

Course and Termination. — **Prognosis.** — Acute peritonitis is very

fatal. Among 100 cases from the London Hospital records (p. 610) there were seventy deaths and thirty cases of recovery.

The recoveries will mostly be found among the cases due to trouble in the appendix vermiformis, among those of pelvic peritonitis, and those of localised peritonitis in the subdiaphragmatic region. Of the thirty examples of recovery in the above-mentioned series of cases, sixteen were instances of disease starting in the appendix, and seven examples of pelvic peritonitis.

Other things being equal, peritonitis is most fatal at the two extremes of life; namely, in infants and in advanced age. The most fatal forms of peritonitis are those which depend upon perforation, upon strangulation of the bowel, upon operation, and upon puerperal affection. The mortality in all these varieties is very high.

Lung complications are common in peritonitis. Among the hundred cases there were no less than seventeen cited in which pleurisy or pneumonia appeared after the peritonitis had set in. The right side of the chest is more commonly affected than the left.

Of the seventeen cases, seven were in association with peritonitis of intestinal origin, six with pelvic peritonitis, and four with peritoneal inflammation following wound.

As to the time of death in peritonitis, the following is the result of an analysis of the seventy cases mentioned :—

In 33 per cent of the examples death occurred between the fifth and the seventh day. In 27 per cent death took place after the seventh day; in 20 per cent between the third and fifth day, in 6 per cent within forty-eight hours, and in 14 per cent within thirty-six hours. It is needless to say that the most rapidly fatal cases are in association with perforation of the bowel. Death is very rapid also in some cases of puerperal peritonitis.

Of the cases which ended in recovery there was in some more or less complete resolution: in others the peritoneal inflammation became subacute or even chronic, and in the remainder a definite localised collection of pus appeared.

Examples of cases ending in recovery are provided by certain forms of perityphlitis.

Diagnosis.—From what has been said already it is evident that acute peritonitis does not always follow a peculiar course, and that, if certain symptoms are insisted upon as essential to a diagnosis, there may be a difficulty in forming a correct opinion.

In not a few instances the peritonitis appears as a late event in the course of typhoid fever or intestinal obstruction, and its manifestations are possibly obscured by the symptoms which have preceded it.

In certain conditions, to which allusion has been already made (p. 623), the clinical manifestations may be masked, and again, under the influence of morphia, some of the characteristic phenomena may be almost blotted out.

We have seen that a common train of symptoms ushers in most of the really acute lesions within the abdomen, and that for some hours, at least, it may not be possible to say whether the case be one of renal or hepatic colic, of perforation of the bowel, or of twisting of an ovarian pedicle. In a little time, however, differentiating signs appear, and the diagnosis can usually be made within more or less narrow limits, or indeed with absolute precision.

It is hardly necessary to dwell upon the differential diagnosis of acute muscular rheumatism of the abdominal wall, of acute pleurisy or pneumonia, or of certain phases of hysterical mimicry. These affections are still mentioned by authors as conditions likely to give rise to error in diagnosis; some also add peritoneal hæmorrhage and diabetic coma. The hæmorrhage may produce the symptoms of acute peritoneal damage which have been described under the title of "Peritonism" (p. 616).

Two maladies, however, need some consideration in connection with the differential diagnosis; namely, colic and intestinal obstruction.

Colic.—The collection of symptoms known as colic may be illustrated by the colic of lead poisoning. In acute examples this colic may bear some resemblance to peritonitis; certain points of distinction, however, have to be noticed. The pain in colic is distinctly paroxysmal; the patient recognises it as a griping pain, it bends him double, he tends indeed to assume a position which is the very opposite of that assumed in peritonitis. There is, as a rule, no hyperæsthesia of the surface and no tenderness; often, indeed, the pain is relieved by pressure. There might be some collapse in severe colic, but vomiting is rare. There is no meteorism and no fever, although now and then cases of lead colic are met with in which there is tenderness of the surface, with inability to bear pressure and with fever. Intestinal movements take place in colic, and can often be perceived both by the patient and the medical man; the patient rolls about, and keeps his hands pressed to his abdomen. There is a total absence of the phenomena either of inflammation or of septicæmia. I think, however, that sometimes at the outset of peritonitis the pain may be in fact the pain of colic.

Intestinal obstruction.—In acute intestinal obstruction the symptoms at the onset are those of "peritonism." The pain is more of the nature of colic; the patient often enough is bent double. The pain is disposed (at first at least) to be paroxysmal. The abdomen is not tender, nor is the belly wall rigid and board-like. Meteorism appears early. Vomiting is an early symptom, is frequent, and very copious. It often gives a temporary relief, and in a little while in acute cases the vomited matter becomes stercoraceous. Abdominal respiration is not inhibited at first. There is no fever.

Treatment of acute peritonitis.—The following are the principal elements in the treatment of this affection:—

(i.) *Rest.*—Absolute rest in the recumbent position appears to be the first obvious indication. The knees may be flexed over a pillow to lessen

the tension on the abdominal walls, and to favour the patient's instinct to assume that posture. The upper part of the chest and the ever-restless upper limbs should be protected by a woollen jacket, and no reasonable restraint offered to the patient's inclination to hold the hands above the head. This favourite posture, by acting upon the lower part of the thoracic wall, assists also in diminishing the tension within the abdomen. It is cruel to insist that the hands shall be kept beneath the bed-clothes. The misery of restlessness has a natural relief in little movements.

(ii.) *Feeding.*—The old rule of eighty years ago of giving as little food as possible by the mouth cannot be bettered. The stomach is not in a state to receive nourishment, and what is taken usually remains unutilised and is returned unchanged. The practice of laboriously plying the patient with teaspoonfuls of this meat extract or of that recalls the legend of the Danaïdes who spent their energies in pouring water into sieves.

In the matter of feeding two extremes are to be avoided: the first is the rigid, unreasoning, and often needlessly cruel prohibition of food of any kind by the mouth, and the second is the reckless and intemperate use of ice or iced fluids.

Thirst is often a most terrible symptom in peritonitis. It is not always quite relieved—at first, at any rate—by rectal injections. The patient is ready to give his life for a drop of cold water. If he takes it he is sick, but he is much relieved. This little indulgence does not introduce anew the symptom of vomiting. It is there, but it is latent, and the drop of cold water only makes it evident. The patient will not die of vomiting, and if a rigid exclusion of all fluid by the mouth renders the symptom latent, it does not abolish it. The man would sooner vomit than endure his thirst. Very often great relief is given by allowing a fairly copious draught of fluid, which is soon rejected, and which—as it were by washing out the stomach—leaves the patient for a while infinitely comforted. On the other hand, the perpetual sucking of ice is as bad as the perpetual teaspoonful of useless jelly or of decomposable meat juice. The filling of the stomach with iced water does undoubtedly add to the general depression of the patient, especially when vomiting has been brought into abeyance by morphia.

The right course appears to lie between these two extremes. There should be no rigid formula except this—let the patient take as little food as possible by the mouth. If there be a raging thirst, let him have a little ice—and very little suffices—or a little iced milk and soda-water; or if, as is common, his inclination lie towards something warm, let him take a few spoonfuls of hot water or of weak tea made hot, or of beef tea at a like temperature. It is not the nourishment that is needed, but some fluid in the stomach.

As regards actual feeding — in the usual sense — that should be carried out by rectal injections. The injections should be small in amount, should be weak, should be made of some peptonised meat extract

or infusion, should be given slowly, and should be of the temperature of the body.

An injection of two to two and a half ounces of peptonised beef tea with half an ounce of brandy appears to answer well, and may be given every three or four hours. Thirst is better relieved by an injection of half a pint of warm water given alone, than by diluting the nutrient enema to that extent. I do not think that nutrient suppositories are so well suited for cases of peritonitis as is the ready-made fluid injection. Irritability of the rectum may be met to some extent by washing it out now and then, and by a weak injection of cocaine. In the more advanced cases the sphincter begins to relax as the loss of power progresses, and then very usually nothing can be retained. It is undesirable to vex the last hours of a dying man by nutrient enemas simply because it is the custom.

If there be diarrhœa, or if the bowels be acting freely, then rectal feeding cannot be adopted; but, fortunately, in these cases the vomiting is as a rule slight, and fluid and a certain amount of nourishment can be taken by the mouth. It is, perhaps, needless to add that in cases—with or without diarrhœa—in which there is no vomiting, all that is given may be given by the mouth.

Much complaint is often made by patients with peritonitis of the horrible state of the mouth. The tongue is dry and stiff, and such sense of taste as remains is only capable of perceiving a bitter nastiness. Something can be done to relieve this by keeping the teeth brushed, by washing the mouth out with a 1 in 80 carbolic lotion or with a mixture of eau de Cologne and water, by directly cleaning the tongue, and by keeping it moist with glycerine and water.

(iii.) *Opium.*—Here, again, the old practice appears to be the best. Give as little opium as is possible. In the early stages of acute peritonitis, and especially in the perforative forms and in those depending upon appendix troubles, morphia in the hypodermic form is absolutely necessary. In the worst instances it may certainly avert death from shock. Under its influence the patient revives, and the more intense symptoms become greatly modified.

Morphia should never become routine practice in peritonitis. It masks the symptoms, hinders the natural process of cure, and hampers treatment. The indication for it is actual pain, not mere restlessness and misery. In the septic forms but very little morphia is called for, often none at all. Its evil effect in the after-treatment of cases of abdominal section has been amply demonstrated. In quite hopeless cases there can be no objection to its freer employment, but in others the administration and the dose of the drug must be sanctioned and measured by the one symptom, pain.

I have noticed that in those who are dying of general peritoneal sepsis, a greater sense of relief usually follows the hypodermic injection of strychnia than of morphia. The strychnia appears to act as a stimulant —the patient revives for a while and feels more hopeful under its

influence, his moanings cease, his miseries are less unendurable, and for the twentieth time he thinks he may get better.

(iv.) *Aperients.*—At the beginning of the century the use of aperients was a necessary element in the routine treatment of peritonitis. By aperient treatment may be understood the obtaining of an action of the bowels by either drugs or enemas. After a certain number of years the practice altered, and the dictum went forth that when any signs of peritonitis were present aperients were to be absolutely forbidden.

Within the last few years the more ancient method has been in a small degree revived, but it has been revived with very radical modifications. To Mr. Lawson Tait the profession is indebted for this return to an almost forgotten practice, and, above all, for the employment of that practice with the new element of discrimination. Mr. Tait's measure has been frequently spoken of as "the treatment of peritonitis by aperients," and it has been assumed by some—and probably with disaster—that a purgative is necessary in every case of peritoneal inflammation. Mr. Tait's precise words on this subject are as follows:—"I have never said that the purgative treatment will cure peritonitis, for peritonitis, once it is completely established, is a practically incurable disease and almost uniformly fatal" (16).

It is on this very point that the centre of the position with regard to this treatment turns. Aperients can never be adopted in the routine treatment of peritonitis. In the larger proportion of cases this measure is entirely useless, and in the great series of the septic forms it is more or less impracticable. In most of these septic examples enemas and purges of any kind may be administered, but the probability is that the bowels will not act at all, and if they do it is more than probable that the treatment will not affect the prognosis in the least, certainly not in the patient's favour. If the aperient could eliminate the fatal poison which is circulating in the patient's system then good might follow, but even the most enthusiastic advocates of purging cannot credit their drugs with this power.

No doubt there is within the intestine an amount of noxious or poisonous matter which remains harmless so long as the viscus retains its normal condition, but which may lead to septic symptoms if certain changes are induced in the wall of the bowel or possibly in its contents. This has been especially shown in connection with the potentialities of the colon bacillus, and it is clear that these noxious elements include not only simple chemical substances, but also various micro-organisms and their hurtful products. It appears to be equally evident that the injurious matters within the disordered bowel, within certain possibly narrow limits, can be got rid of by the action of aperients.

The condition in which aperients appear to be especially useful is in that called by Olshausen "pseudo-ileus." This name is applied to certain symptoms which are sometimes met with after abdominal operations—and possibly under other circumstances; and which may certainly lead

to actual peritonitis. They are not so much the phenomena of any form of peritonitis as of a condition which may precede it.

The symptoms are these:—On the second or third day after a not necessarily severe abdominal operation the abdomen becomes distended, the patient becomes uncomfortable, and complains of "wind." The distension increases; vomiting sets in. At first only the contents of the stomach are rejected; very soon the matter becomes bilious, and finally there is copious "coffee grounds" vomiting, and the vomit may present a fæculent odour. The vomiting tends to become worse and worse, and is much more copious than is common in peritonitis. The pulse becomes smaller and feebler, the temperature usually sinks, and exhaustion increases with alarming rapidity. On the fourth or fifth day after the operation the patient may die. At the necropsy the peritoneum may be found practically unchanged, or to exhibit so trifling a degree of peritonitis as not to account for the symptoms nor for the fatal result. Various explanations of this condition have been given. There is no doubt that the nervous disturbance which attends any abdominal operation leads to some degree of intestinal paralysis. This paralysis, attended as it is by vaso-motor changes in the bowel wall, is favourable to the absorption of septic matters from the intestine. The paresis may be slight and may disappear spontaneously, or it may subside if the distension of the bowel can be relieved by the introduction of a rectal tube. If it persist it appears to permit of a filtration of septic materials, of bacteria, or of their products, through the intestinal wall. These agents are readily taken up by the peritoneum and a septicæmia commences.

Anyhow, it was pointed out by Mr. Tait, and has been made evident to most surgeons who have followed his teaching, that if an action of the bowels can be obtained at the outset of the symptoms, either by the administration of a purgative or by an enema, the trouble in a large proportion of cases passes away and the patient makes a good recovery. It cannot be said that this good result follows in all cases, and it is evident that the purgative, like the emetic given in acute poisoning, can only bring about an arrest of the symptoms within certain limits. When general peritonitis has once established itself an aperient is unavailing. In those septic cases in which diarrhœa occasionally sets in this is only too apparent. In some examples of perityphlitis the prompt evacuation of the bowels is often attended with the very best results.

(v.) *Blood-letting.*—This measure of treatment may with advantage be more extensively employed. It is no longer likely to be used in the unreasoning and mechanical fashion of bygone days. In robust forms of localised peritonitis blood-letting is attended with admirable results. In perityphlitis, again, the application of half-a-dozen leeches often acts with magical effect. In the older accounts of the treatment of peritonitis by bleeding no good appears to have followed in cases in which the inflammation was diffused, except, perhaps, in some examples due to injury. On theoretical grounds this is precisely what would be expected.

(vi.) *Operative measures.*—These are represented by incision and drainage, with or without irrigation. This treatment must be considered as it applies to peritonitis under two entirely different aspects. In one series of cases there is vigorous, well-defined inflammation, the local symptoms are marked, pus is produced and may be considerable in amount, and the exudation is more or less clearly localised. Examples under this heading are afforded by peritonitis started by mischief in the vermiform appendix, by many forms of peritonitis within the pelvis and in the subphrenic region, and by certain cases of limited inflammation following injury or perforation.

In the other series of cases the peritonitis is diffused, the constitutional symptoms are more prominent than the local ones, the changes in the serous membrane—so far as evidence of inflammation is concerned—are comparatively slight, and are out of proportion to the general disturbance. This form is illustrated by cases in which there is a general septic intoxication starting from the peritoneum, by peritonitis due to perforation or following after strangulated hernia or enteritis, by puerperal peritonitis, and by examples of genuine peritonitis following operations upon the abdomen.

In the first series of cases surgical interference by incision and drainage ranks with the procedure of evacuating a large abscess.

In the second series the cut into the abdomen and the subsequent flushing out or drainage are to be compared with the washing out of the stomach after an active poison has been swallowed.

In the one case the body has to be rid of the products of a robust and possibly limited inflammation; in the other case an attempt has to be made to remove from a cavity a poison which has already wrought no little harm. The operation in the latter instance is directed not so much against an inflammatory outbreak as against a progressive poisoning.

The operative treatment of suppurative peritonitis, especially when the effusion is localised, has been remarkably successful. Records of the operation extend back into the eighteenth century, and all that modern surgery can lay claim to is the application of the treatment with greater boldness, with greater frequency, and with infinitely less delay.

The operative treatment of general diffused non-tuberculous peritonitis has little progress to chronicle. When once this diffused peritoneal inflammation is well established the possibility of saving life by operation is not great. The measure which has met with most success is represented by the opening of the abdomen, by the free evacuation of the peritoneal effusion, and finally by the evacuation of the bowel by means of a temporary fæcal fistula established in the small intestine. That the bowel should be emptied in the majority of these cases is most desirable. Operation to be of service in the acute and diffuse form of peritonitis must be done early. If the gravity of the patient's position be appreciated, it will be evident that a carefully made incision into the abdominal cavity can hardly add to his peril, while on the other hand it may be the means of saving his life.

The treatment of perforative peritonitis by operation has become less desperate in proportion as the laparotomy has been performed the earlier. There is little to be gained by delay in such cases.

In peritonitis due to intestinal obstruction or to puerperal septicæmia, the results of operative interference have not so far been very encouraging. The prognosis will be less gloomy in these two affections when the former condition is treated by early exploration, and when the latter is prevented by suitable antiseptic precautions.

FREDERICK TREVES.

REFERENCES

1. COURTOIS-SUFFIT. *Soc. méd. des hôp.* April 1890.—2. ESCHERICH. *Fortschritte d. Med.* 1885.—3. GALLIARD. *Soc. méd. des hôp.* Nov. 1890.—4. GUBLER. *Journ. de Thérap.* 1877.—5. HOROWITZ. *Wien. med. Wochens.* 1892, No. 2.—6. LARUELLE. *La cellule*, 1889, vol. v.—7. MACAIGNE. *Le Bact. coli commune.* Paris, 1892.—8. MALVOZ. *Arch. de méd. expér.* 1891, p. 593.—9. MENGE. *Zeits. für Gebrts. und Gynäkol.* 1891, xxi. p. 19.—10. MORISSE, *Péritonite à pneumococcus.* Paris, 1892.—11. NELATON. *Bull. méd.* Oct. 1890.—12. PERE. *Annales de l'Institut Pasteur*, July 25, 1892.—13. REICHEL. *Deutsche Zeit. für Chir.* Bd. xxx. p. 1.—14. SEVESTRE. *Soc. méd. des hôp.* April 1890.—15. SPILLMANN and GANZINOTTY. "Péritonite," *Dict. encyc. des sc. méd.* Paris, 1887.—16. TAIT, LAWSON. *Brit. Med. Jour.* Nov. 12, 1892, p. 1050.—17. TAVEL and LANZ. *Ueber die Aetiologie der Peritonitis*, Basel, 1893.—18. TERRILLON. *Progrès médical*, 1883, p. 645.—19. WEICHSELBAUM. *Central. für Bakt.* Jan. 1889.—20. WELCH. *Trans. Congress of Amer. Phys. and Surg.* vol. ii.—21. WERTHEIM. *Central. für Gynäkol.* 1892, No. 20.—22. WURTZ. *Arch. de méd. expér.* Jan. 1, 1893.—23. YEO, BURNEY. *Brit. Med. Jour.* vol. i. 1894, p. 1289.

F. T.

ACUTE PERITONITIS OF UNDETERMINED NATURE

I. Peritonitis associated with erysipelas.—That the various serous membranes of the body are occasionally the seat of acute inflammation in the course of erysipelas is generally recognised; and having regard to the relationship of these structures to the lymphatic system, and to the spread of the virus of the infective process by the lymphatic channels, the coexistence might perhaps be expected to happen more often than it does. The infrequency of the implication of the peritoneum is perhaps explained by the fact that the lymphatics of the abdominal wall do not *directly* communicate with it.

Excluding from present consideration the cases of peritonitis associated with erysipelas which occur in the puerperal state (see Puerperal Peritonitis), and those in the new born (*vide infra*), authentic records exist of the occurrence of peritoneal inflammation secondary to extensive erysipelas of the trunk, and more particularly of the abdominal parietes (36, 8).[1]

[1] Dr. Wilks records the case of a child aged two years who died a month after an extensive burn of the abdomen. Two days before its death severe symptoms set in, and acute general peritonitis was found after death. The occasional concurrence of duodenal ulcer with both burns and erysipelas may be remembered in this connection.

The clinical histories of such cases, however, give but little evidence of symptoms referable to the peritonitis, and sometimes no suspicion of the complication has existed, post-mortem examination alone revealing the condition of the serous membrane. Pain and tenderness, when present, appear to be but slight, and are difficult to separate from these same symptoms due to the erysipelas; some vomiting has been noted, and a fall rather than a rise of temperature has been said to mark the onset of the peritonitis. So far as is known, the condition is always fatal within a very few days. After death a general acute peritonitis is found, marked by much hyperæmia of the vessels, stickiness of the surface of the membrane, and a variable, though inconsiderable, amount of purulent effusion, with flakes of fibrinous lymph lying among the slightly adherent intestinal coils.

II. Peritonitis in connection with certain eruptive fevers.—Among the rarer complications and sequels of the exanthems, acute peritonitis must be mentioned along with acute pleurisy, pericarditis, and meningitis, with one or more of which it occasionally coexists. Infrequent as it is, it would seem, like other inflammations of serous membranes, to be met with somewhat oftener in *scarlet fever* than in other maladies of this group, and in this disease it may be independent of a nephritis, or associated with it. In *measles* and *small-pox* acute peritonitis has been seen, but most exceptionally. In *typhus fever* it has occurred with scarcely greater frequency; in one case (9) the onset followed a cold bath, administered to reduce the temperature, and in another (24) it was obviously attributable to the rupture of a splenic infarct, the result of endocarditis (21). To the last-mentioned accident, or to dysentery, are to be ascribed the rare cases of acute peritonitis which complicate *relapsing fever*. In *enteric fever* peritonitis is not uncommonly a result of intestinal perforation, less often of a ruptured mesenteric gland, and is sometimes caused, as it would seem, by the passage of septic matter from the ulcerated mucous surface through an intact peritoneal coat. Under all circumstances the complication is an exceedingly fatal one, though recovery is not unknown. For the most part the symptoms are but slightly marked, unless the determining factor be perforation of the bowel.

Among the many sequels of *influenza* acute peritonitis is to be numbered.

III. Epidemic peritonitis.—An acute peritonitis has on rare occasions been known to prevail in a number of cases so considerable, within a time so limited, and over an area so restricted, as to warrant—at least provisionally—the application of the term *epidemic*.

Excluding the doubtful examples of such a condition reported by some authors as having occurred in the army early in the century, the best known account, though in some respects incomplete, is that of the outbreak which took place in August and September 1874, at the Royal Victoria Patriotic Asylum for girls at Wandsworth, in the investigation of which Dr. Anstie lost his life. Fifty-two cases occurred, and of these four were fatal. The outbreak had been preceded by an epidemic of "catarrhal fever," and by erysipelas in a few of the children.

A careful examination left little doubt that the drinking of rain water which had become foul, partly from contamination by sewer gas, was an immediate cause. Such are all the facts now known; no clinical records, or records of the post-mortem appearances in the fatal cases, are available. It was argued at the time that the catarrhal fever (which was a malady attacking the respiratory tract, commencing with sore throat, followed by an eruption about the lips) was a cause of the erysipelas, and that this latter in some unexplained way determined the peritonitis (11).

Scarcely more complete is an account of an outbreak in the province of Ontario, Canada, in the winter of 1886, which is recorded by Dr. James Samson. During a period of ten months thirty cases were seen within a radius of four or five miles, and of these eleven died, mostly within two days. The persons attacked were previously in good health; and, in those which recovered, convalescence did not set in for a fortnight from the beginning of the illness. Severe epistaxis was noted in most of the cases.

A very similar epidemic in a neighbouring district was described by Dr. Dewar in the following year.

IV. Rheumatic peritonitis.—The very existence of the affection is disputed by some, and regarded as more than doubtful by many of the most recent and highest authorities (7). Until the exact nature of rheumatism is itself satisfactorily explained, and the precise range of morbid states comprised within this name ascertained, much difference of opinion concerning inflammation of the peritoneum as a rheumatic manifestation is likely to prevail. On general grounds a rheumatic peritonitis would appear to be highly probable, since inflammation of the serous membranes of the joints and of the pericardium are among the most frequent "terms" of the "rheumatic series"; somewhat less commonly a pleurisy, and still more rarely a meningitis are met with in the course of an acute rheumatic attack, but fewer still are those cases of peritonitis which appear to have had the best claim to be regarded as rheumatic (*vide* references Nos. 27, 33, 30, 35, 13, 26). No explanation at present is forthcoming for this remarkable freedom of the peritoneum.

It is certainly significant that in the greater number of cases recorded there was a coexistent pleurisy, and frequently a pericarditis or pneumonia, or both; and the possibility of extension of the inflammation from the pleura to the peritoneum must not be lost sight of in determining the specificity of the rheumatic origin. After excluding all doubtful cases, however, there still remain some in which a peritonitis of rapid onset, transient duration, and equally rapid disappearance (features which are eminently characteristic of an acute rheumatic course), appeared either before well-marked arthritis and other accepted rheumatic phenomena, or accompanying them, or after them; such cases seem to justify—provisionally, at least—the inclusion of an inflammation of the peritoneum among the various manifestations of the rheumatic diathesis. A remarkable case is recorded by Martin of a patient who, for nine years, exhibited almost annually some rheumatic symptoms: articular rheumatism,

two attacks; pericarditis, two attacks; muscular rheumatism; cerebral rheumatism (*sic*); and finally succumbed after two days to an attack of peritonitis for which no obvious cause was found.

The *symptoms* as a rule are not well marked, and offer no features of distinction from acute peritonitis otherwise caused; the course is rapid, and a fatal result, for which the affection of the serous membrane is not wholly responsible, is the rule rather than the exception. After subsidence of the symptoms they have been known to recur in the course of the same rheumatic attack. On autopsy the usual appearances of an acutely inflamed peritoneum are observed, together with an effusion which is seldom considerable, is generally sero-purulent in character, and contains numerous flakes of lymph. No bacteriological examination of the effusion has hitherto been recorded, but the obvious characters of the fluid suggest the presence of micro-organisms, the source of which remains for the present a matter of conjecture. It is permissible to suppose, however, in accordance with the views generally accepted, that the microbes are derived from the alimentary tract, and gain entrance to the peritoneal cavity through the walls of the canal which certain morbid changes, induced in this case by the "rheumatic poison," have made less resistant to their passage. But it is quite unknown whether the hypothetical toxic substance acts primarily, by way of the blood-vessels, upon the peritoneum, the changes in the intestinal walls proceeding from the serous covering towards the mucous—whether, that is, a peritonitis primarily toxic subsequently becomes septic—or whether the poison induces its effects in the opposite direction, namely, from the mucous outwards to the serous layer, damaging the tissues and so permitting the migration of the organisms—whether, that is, the peritonitis be septic from the first and secondary to "rheumatic changes" in the viscera. In support of the former hypothesis it has been found experimentally that the injection of chemical irritants into the abdominal cavity of animals may be followed by a sero-fibrinous exudation, free at first from organisms, but in which organisms have subsequently been found with purulent results; the inflamed coats of the intestines having permitted their passage inwards.

The latter view has been strongly advocated by those observers who have ascribed many cases of a localised form of peritonitis, namely, perityphlitis, to rheumatic agency. Noting the very large amount of lymphoid tissue which enters into the structure of the appendix vermiformis, and the close similarity the organ bears in this respect to the tonsils, and having regard to the frequency with which acute inflammation of these bodies is associated with other rheumatic manifestations, they are disposed to consider a rheumatic appendicitis as bearing the same relation to the underlying constitutional state as does rheumatic tonsillitis, and thus to consider the perityphlitis as secondary to the affection of the appendix. Should this ultimately prove to be a true explanation, a stronger justification for the existence of a "rheumatic peritonitis" will be forthcoming than exists at present. Clinically, however, the evidence for rheumatic perityphlitis is far from being complete; to a great extent it is based on

the fact that certain patients, who presented symptoms of appendicitis, recovered on treatment by salicylate of soda (37, 4, 31, 22, 10). This kind of argument is not quite satisfactory, and is insufficient to warrant a belief that "gouty perityphlitis is extremely common," or that, "in other words, perityphlitis nearly always owns this origin, and that if all cases were treated by salicylates at the outset, further troubles and surgical procedures would be rare" (17). The frequent recurrence of attacks of appendicitis is alleged in support of the hypothesis of a constitutional (rheumatic) origin for the disease, for which it is said to account more satisfactorily than the more mechanical causes to which it is usually attributed.

V. In association with nephritis.—An acute general peritonitis is not an uncommon complication of Bright's disease, whether acute or chronic, perhaps more often of the latter, and especially of the chronic interstitial variety; but, as is the case with rheumatism, the peritoneum is much less frequently affected than are the pericardium or pleura (19). Inferences drawn from the statistics of various authors cannot be very precise, inasmuch as no distinction is drawn between the various forms of nephritis in regard to the incidence of peritonitis; but the complication probably occurs in about 10 to 12 per cent of all cases of Bright's disease. Occasionally in these cases the peritonitis coexists with a pleurisy or pericarditis.

The symptoms are even more apt to be in abeyance than they are in the rheumatic state, and the condition is often unexpectedly discovered after death. The effusion is purulent, and a marked absence of injection of the serous membrane has been often noticed by Fagge.

The pathology of this condition is obscure, but would seem to be allied to that of "rheumatic peritonitis." The primary irritant is assumed to be some toxic material derived from the products of tissue waste which the diseased kidneys fail to eliminate, and this, like the rheumatic poison, may be supposed to cause a simple peritonitis in the first instance, which in time, as organisms invade the serous cavity from the intestines through their damaged walls, becomes purulent; or, since affections of the intestinal mucous membrane, even to the extent of ulceration, are known to occur in the course of Bright's disease, the peritonitis may be really infective from the first. Extension from the pleura and pericardium may explain some cases. Bauer, after observing that peritonitis is "comparatively frequently observed" in chronic Bright's disease, continues:—"Whether this increased tendency to peritoneal inflammation in chronic degeneration of the kidneys is to be ascribed to the altered constitution of the blood, or to the mechanical element of distension of abundant serous infiltration and stagnation of blood due to the presence of that ascites, cannot yet be determined; for both views there exist reasons for and against. We generally find the peritoneum thickened and swollen after long-standing ascites; nevertheless a terminal peritonitis is more frequently met with in cases of Bright's disease than in dropsy from any other cause."

VI. Idiopathic.—Included within this group are those cases which, though doubtless directly dependent upon bacterial invasion, are not connected with any of the more distinctly defined infective processes already mentioned; nor, in the complete absence of any rheumatic symptoms, are yet to be accepted as "rheumatic" in character. Whether a more precise knowledge of the nature of rheumatism, and especially of its relation, if any, to "chill," will ultimately allow them all to be placed in the same category remains to be seen; but meanwhile they offer sufficient clinical characteristics to justify their being placed in a group apart, and provisionally described as "idiopathic," or "primary acute peritonitis," for lack of a more correct name. Two classes of cases are here referred to.

In the one class adults are the principal sufferers, in the other children between the ages of five and twelve years, mostly girls, (fifteen out of twenty-five). Of the latter class a most instructive and complete case is described by Dr. S. West, which proved fatal in five days, and was marked by a persistently low temperature. Mr. Watson Cheyne has drawn attention to the marked fatality of acute inflammation of serous membranes characterised by low temperatures and associated with streptococcus infection. Mr. Southam's case of acute suppurative peritonitis was apparently idiopathic. In the recorded cases no cause whatever can be assigned, nor can the determining factor of the bacterial invasion of the peritoneum (unless it be by way of the genital tract in some of those occurring in women) be recognised, unless it were exposure to cold, getting wet, lying face downwards on the damp grass, swallowing iced water when overheated, and the like. According to recent observations it seems possible for bacteria to migrate through the wall of the bowel without any obvious lesion, though the very fact of their doing so implies some diminution in the normal resistance of the various tissues concerned. This supposition would go some way to explain many of the so-called idiopathic cases, yet it would still leave unanswered the question, "What determines the greater vulnerability of the coats of the intestine?"—to which "the conditions brought about by chill" is a possible answer. The patients have been almost invariably in perfectly good health previously, the first symptoms complained of have generally been referred to the abdomen, and, with a few exceptions in which empyema has supervened, they have been confined to the same region. The character of the attack is most acute and severe, causing death generally within a few days. The abdominal pain and tenderness, the vomiting, constipation and meteorism, small quick pulse with anxious countenance, anorexia, thirst and high temperature correspond to the ordinary features of acute peritonitis in its fulminant form.

Such cases are apt to be mistaken during life for peritonitis due to perforation of the stomach or intestine, or for acute intestinal obstruction; and, as on necropsy no cause for their occurrence is revealed, their diagnosis and their pathogeny are equally obscure. The limitation of the symptoms and the restriction of the morbid changes in almost every case

to the peritoneum, is strikingly in contrast with those cases already described as rheumatic, and suggest a comparison with those not infrequent examples of primary pleurisy, simple and purulent, for which no more conclusive cause can be alleged than "chill." Dr. Foxwell, however, records a case of a man aged fifty, and another of a child aged four years, both of whom died, and in both after death an acute purulent peritonitis was found together with some pus in each pleural cavity in the man, and a pint of pus in the pericardium of the child. No history of rheumatism or of other cause is given. The comparison becomes still closer if we include cases of acute primary ascites, such as are occasionally described, in which the effusion is wholly serous. Nevertheless, similarity, even if valid, does not furnish an explanation.

In connection with the so-called idiopathic peritonitis, certain observations of Dr. H. P. Hawkins are not to be forgotten. Dr. Hawkins in certain cases of this kind made microscopic sections of the appendix which to the naked eye appeared quite normal. The microscope, however, in the cases examined, revealed minute abscesses in its structure; one of them by bursting into the peritoneal cavity might set up a peritonitis, apparently "idiopathic."

Laparotomy, with flushing and drainage of the peritoneal cavity, offers the best prospect of relief.

PERITONITIS OF THE NEW BORN.—Peritonitis, as it is met with in the new-born infant, occurs both in the acute and in the chronic form. The latter is almost always the result of prenatal syphilis, and will be considered hereafter.

The acute cases owe their origin to two very different causes; in the one group, which is becoming much less numerous as more exact precautions are observed, the infection of the peritoneum follows the absorption of septic matter, due to the ligature and separation of the umbilical cord; but Runge has shown that in the puerperal septicæmia of the new born peritonitis is much less common than pleuro-pneumonia; four cases only occurred in fifty-five infants dying of the general disease. Such cases are invariably fatal within a week. Associated with the peritoneal affection, and perhaps secondary to it, an erysipelatous condition of the abdominal walls has been met with.

In the other cases rupture of the colon, especially in the neighbourhood of the sigmoid flexure, has been found, the intestinal lesion being attributed to pressure on the distended abdomen during parturition. This explanation cannot be regarded as satisfactory, for no such condition is met with in imperforate anus, when the resistance of the meconium filled bowel would be far greater. A singular case of localised peritonitis, evidently of some duration, and fatal the day after birth, is described by Generisch; it was apparently due to perforation of a Meckel's diverticulum, or to non-closure of the opening of the vitelline duct.

In two cases of infants (6), aged twelve and fourteen days, a fatal peritonitis occurred for which no cause could be assigned, unless it were

coexisting intestinal catarrh. The inflammation was circumscribed, and chiefly in the neighbourhood of the ascending and transverse colon.

The recognition of this condition during life is not easy; the evidence of abdominal pain and tenderness at this age is very indefinite, and vomiting and constipation are less marked than in older children; moreover there is a proneness to cerebral symptoms which obscure the diagnosis.

Treatment must be preventive; the disease once established is fatal.

W. H. ALLCHIN.

REFERENCES

1. ASHBY. Keating's *Cyclopædia of Diseases of Children*, vol. iii. 1890.—2. BAKER, M. *Clin. Soc. Tr.* xix. 1886, p. 43.—3. BESNIER. *Dict. encycl. des sc. méd.* art. "Ascite."—4. BRAZIL. *Brit. Med. Jour.* May 25, 1894.—5. BREMEN. *Annuel de gynécologie*, Jan. 1890.—6. CASSEL. *Berlin. klin. Woch.* Oct. 17, 1892.—7. CHARCOT, BOUCHARD, and BRISSAUD. *Traité de médecine*, vol. v. p. 533; 1893.—8. CHEURLIN. *Thèse de Paris*, 1879.—9. COLLIE. *Lancet*, 1872, 16th Nov.—10. *Edin. Hosp. Rep.* 1895.—11. *Fourteenth Report of the Royal Commissioners of the Patriotic Fund*, 1875.—12. FOXWELL. *Essays in Heart and Lung Disease*, 1895, p. 164.—13. *Ibid.* p. 161. 14. *Ibid.* p. 158.—15. GAUDERON. *La péritonite idiopathique aigue*, Paris, 1876.—16. GENERISCH. *Virchow's Archiv*, 1891, Bd. cxxvi. p. 485.—17. HAIG. *Practitioner*, Jan. 1893; *Brit. Med. Jour.* June 30, 1894.—18. HAWKINS, H. P. *St. Thomas's Hospital Reports*, 1892.—19. HENOCH. *Diseases of Children*, Sydenham Society Translation, 1889, vol. ii. p. 149.—20. HOROWITZ. *Wien. med. Woch.* 1892.—21. JENNER, Sir WM. *Med. Times and Gazette*, 1850, vol. ii. p. 408.—22. *Lancet*, Aug. 24, 1895.—23. MARTIN. *Gaz. des hôpitaux*, 1873, p. 276.—24. MURCHISON. *Continued Fevers*, 1884, p. 212.—25. PALTAUF. *Virchow's Archiv*, Bd. iii. p. 461.—26. ROBINSON. *Brit. Med. Journ.* 1887, ii. p. 828.—27. RONDOT. "Péritonite aigue generalisée primitive," *Thèse de Paris*, 1878.—28. SAMSON, JAMES. *The Medical Age.* Detroit, Feb. 25, 1894.—29. SOUTHAM. *Medical Chronicle*, July 1892, p. 217.—30. SPILLMANN and GANZINOTTY. *Dict. encycl. des sc. méd.* art. "Péritonites," p. 310.—31. SUTHERLAND, G. A. *Brit. Med. Jour.* 1892, p. 857.—32. TILLNER. *Virchow's Archiv*, Bd. xcvi. p. 307.—33. VIVANT. "Péritonite rhumatismale," *Thèse de Paris*, 1884.—34. WEST, S. *Clin. Soc. Tr.* xix. 1886, p. 36.—35. WHITE, HALE. "Case of Inflammation of several Serous Membranes," *Trans. Clin. Soc.* xix. p. 296; 1886.—36. WILKS. *Guy's Hospital Reports*, 1861, p. 175.—37. YEO, BURNEY. *Brit. Med. Jour.* June 16, 1894.

W. H. A.

CHRONIC PERITONITIS

Etiology.—The conditions with which chronic peritonitis is found associated, and which in varying degree may be regarded as causal, may conveniently be arranged in two groups: (A) Cases following an acute peritonitis, in the etiology of which their causation is involved. (B) Cases which, from the beginning, have run a chronic course, occasionally manifesting acute or subacute outbursts.

A. As with most other diseases which pass from an acute into a chronic condition, no very definite point can be taken from which to date the latter characters. But when on the subsidence of an acute attack of peritonitis the temperature falls, and pain, tenderness, vomiting

and other characteristic symptoms abate, yet complete recovery does not take place, and other and in the main less definite symptoms supervene, then the case is said to become chronic. In an acute case which has completed its course, and ended in a total absence of symptoms and in restoration to health, the products of the inflammatory process either become broken down and removed by absorption or, as often happens, membranes and fibrous adhesions are found as the direct results of the acute condition. On examining the abdomen in later years these adhesions are often wrongly regarded as evidences of chronic peritonitis—the more especially since, as will presently be seen, similar structures do no doubt form an important part of the changes brought about by the chronic affection. Between the events of an acute or of a chronic peritonitis no very distinct line can be drawn, and hence on mere anatomical appearances cases of the acute disease lapsing into the chronic are likely to be counted as more numerous than the clinical histories would actually warrant. We must avoid any confusion between the static results of past peritonitis and the slowly progressive changes of the chronic disease.

Without doubt the acute cases which are the most likely to pass into a chronic state are those in which the affection is circumscribed, and especially if there have been several attacks; the encysted collection of inflammatory products giving rise to a slowly spreading inflammation which tends to involve the entire serous membrane. It is not infrequent even for the most chronic cases to be marked by temporary exacerbations of an acute or subacute character, as if indeed the slowly progressive malady were itself an excitant to outbreaks more or less acute.

What may be the immediate irritant determining the chronic inflammation in these cases is not exactly known. The excitants of the acute condition from which they have lapsed were certain species of micro-organisms, and it is probable that the chronic changes which may subsequently ensue are due either to the irritation induced by some of the inflammatory products, or to such a perversion of the formative nutrition of the affected tissues by the acute disease that they no longer maintain their normal structural integrity, but follow a course which results in the production of those tissue changes which characterise the inflammatory process.

B. The cases which are unattended at their outset by acute symptoms, although these may subsequently arise in the slow progress of the disease, owe their causation to a number of conditions, some of which are most obscure, and none of them clearly understood. They may be provisionally grouped as follows:—(*a*) Idiopathic; (*b*) Toxic; (*c*) Chronic congestion of the peritoneal vessels, and prolonged ascites; (*d*) Extension of inflammation from subjacent structures; (*e*) Traumatic; (*f*) Associated with new growths. Such an arrangement is clearly based on no scientific principle, but merely on grounds of passing convenience in order to bring under a few heads the many conditions which may precede the manifestations of the peritoneal affection. Clinically it is more important to recognise whether the attack be primary or secondary, general or localised, than

to establish the cause, which indeed is often far to seek, complex or inexplicable.

(*a*) *Idiopathic.*—Under this designation—admittedly inexact—are included those cases which supervene (α) after exposure to cold and wet, fatigue, or to other ill-defined hygienic errors; (β) those which on rare occasions follow certain of the eruptive fevers, especially measles, precursors which very inadequately indicate the true cause; and (γ) those for which antecedents still more vague may be alleged as the determining factors.

The late Dr. Habershon (6) may be quoted to show the disbelief of some physicians in strictly "idiopathic peritonitis":—"Although peritonitis is spoken of and treated as an idiopathic disease, we do not find that it is so; it is excited by injury to the serous membrane, or by the direct propagation of disease; out of 3752 inspections made at Guy's Hospital during a period of twenty-five years, 501 were instances of peritonitis; but we cannot find a single case, thoroughly recorded, in which disease could be correctly regarded as existing solely in the serous membrane." In marked contrast to these figures are the following:—Tapret found 8 idiopathic cases out of a total of 24; and Vierordt found 15 out of 29: that is, for 23 out of 53 cases no definite cause could be ascertained (16). But these statistics are of very partial value as no uniform basis of tabulation is pursued; and as knowledge becomes more accurate, the tables constructed in successive periods become incomparable.

As our information grows more precise the limits of this group may be reasonably expected to grow narrower, as many of the cases herein included, which at present are ascribed to the uncertain conditions just enumerated, will be assigned to their proper cause. Some observers, indeed, are much inclined to question the existence of these cases apart from tubercle; and it may be admitted that, inasmuch as many of them end in recovery and post-mortem evidence is wanting, distinctive differences are hard to find.

There is, however, an increasing disposition to admit the existence of a true non-tubercular chronic peritonitis, without a clearly defined cause, and comparable to certain cases of pleurisy which run a similar course, and are of equally doubtful origin. By far the most of these are set down to "chill"—cardiac, hepatic, and other visceral diseases being carefully excluded. Sir George Johnson has described them as "subinflammatory," and they certainly are lacking in any very active or severe characters. The condition has been met with both in adults (10) and in children (12), but apparently with somewhat greater frequency among young girls—a circumstance which suggests that the source of the infection may be a vaginitis or vulvitis. Bauer says that "Louis, notwithstanding his valuable work, has brought a certain amount of confusion into the subject, by making the statement that every case of chronic peritonitis is tubercular."

The exact pathological significance of a "chill" is far from clear to us; if it should prove to be identical with what is now vaguely designated

as "rheumatic," then the cases here referred to will find their correct pathogeny. It may be further noted in this connection that in many of those hitherto reported the peritoneum was not the only serous membrane involved; there was at the same time a general disposition for the fibrous tissues to suffer in a manner we are accustomed to associate with chronic rheumatic affections, or with the changes due to chronic gout. The case of a lad described by Fagge (3) well illustrates this: pleura, pericardium, peritoneum, and tunica vaginalis were similarly affected with effusion into their cavities and other evidences of chronic inflammation, all the viscera being healthy and no signs of tubercle anywhere. Such facts strongly suggest some general (constitutional) cause, for which a parallel may perhaps be found in the well-known liability of some persons to suffer from subacute or chronic inflammation of the mucous membranes, sometimes one and sometimes another being affected; very pertinent to this was the question asked by Fagge, "Is there a catarrh of the serous membranes?" (4).

Some writers have also been inclined to recognise a liability of the fibrous tissues generally to undergo chronic inflammatory changes, without special limitation to one organ—a general fibrosis; if so a chronic peritonitis characterised by much fibroid overgrowth might originate in that way (15). Whether such a condition exists apart from syphilis is doubtful.

During convalescence from a febrile condition a chronic insidious peritonitis has been met with. It is perhaps significant that measles, which is so potent as an indirect cause of tuberculosis and which, as a cause of acute peritonitis, is much less common than scarlet fever or erysipelas (*vide supra*), has been noted as the usual precursor of this state. Henoch records a case of a girl aged eleven years in whom such a peritonitis appeared three months after the specific fever had subsided; complete recovery ultimately followed, after repeated tapping and other treatment.

(*b*) *Toxic.*—Rheumatism, Bright's disease, alcohol, lead, and malaria have each been described, in virtue of their toxic character, as causes inflammation of the peritoneum, and it has further been affirmed that they produce the effect by way of a sclerosis of the arterioles of the peritoneum. Little ground exists for some of these statements. The relationship of the two first-named diseases to acute peritonitis has already been discussed. As productive of a chronic affection of the serous membrane they are, so far as frequency of occurrence is concerned, still less efficient; a chronic peritonitis of rheumatic origin is scarcely claimed even by the warmest supporters of the rheumatic hypothesis; and this is the more remarkable when the marked changes of a chronic character in other serous structures is remembered in this disease. In a small percentage of cases in Bright's disease the post-mortem appearances of a chronic change in the peritoneal structures, both local and general, are to be found (9).

It is very difficult to assign to alcohol its precise share in the pro-

duction of chronic peritonitis, since its effect on the serous membrane alone—if any there be—cannot well be ascertained. Like lead and other metallic irritants, like phosphorus and even the mineral acids, alcohol behaves as a "tissue poison" in proportion to the degree of concentration in which it is exhibited (14). Prominent among the morbid changes induced by these agents are the structural changes of a chronic inflammatory character in the smaller arteries, and following these may come a hyperplasia (fibrosis) of the connective tissue of the parts to which they are distributed. Such changes, as we shall see, are among those which characterise a chronic peritonitis; and to this extent alcohol may be regarded as capable of exciting such a condition. But the serous membrane does not experience the immediate effects of the ingested poison as do the mucous membranes of the alimentary canal and the liver; and it does not seem to be as vulnerable as the nervous and muscular tissues which are so prone to suffer from the intoxicant. In the exceptional cases, therefore, in which a chronic peritonitis is found associated with alcoholism, it is practically impossible to say to what extent the state is primary and independent of the associated lesions in the viscera, especially in the liver and kidneys, which of themselves are among the causative conditions of the peritoneal change. As a factor in the production of that form of chronic peritonitis known as perihepatitis, Dr. Hale White has shown alcohol to be most important; but even in this case it is not clear whether the change in the serous membrane does not originate from the underlying liver. [*Vide* art. "Perihepatitis," vol. iv.]

No confirmation of the statement that lead is a cause of peritonitis appears, after careful inquiry among those who have had large experience of the results of this poison, to be forthcoming; and I can find no cases recorded. An almost similar remark may be made concerning malaria; though in this case the liability to frequent and even prolonged congestions of the abdominal viscera might be held to some extent to dispose to the existence of a chronic peritoneal inflammation; thickening of the splenic capsule and adhesions between this organ and adjacent structures are frequently met with in old cases of malaria.

(*c*) *Chronic congestion of the peritoneal vessels and prolonged ascites.*—It sometimes happens that in the course of an ascites, due either to cardiac or to direct portal obstruction, that symptoms suggestive of a mild attack of peritonitis supervene, subsequent to which, perhaps, the effusion becomes absorbed (11). Or it may be that post-mortem indications of the existence of an inflammatory condition are manifest, together with those of a simple serous exudation from venous congestion. It has been the custom, probably without sufficient reason, and certainly without satisfactory explanation, to speak of such a peritonitis as being caused by the ascites. More probable is it that the excitant of the inflammation is derived from the liver, which is involved to a greater or less extent in the production of the ascites, and has itself undergone considerable change. Such cases, therefore, infrequent as they are, probably belong to the group of causes which follows.

(*d.*) *Extension from subjacent structures.*—The starting point of a chronic peritonitis is not infrequently some morbid change in the parietes or other structures covered by the serous membrane. Reference has been already made to this as the probable explanation of the peritonitis said to arise in connection with the old-standing ascites due to protracted venous congestion. The visceral changes may be the result of injury (to be considered under a subsequent heading), or may be of an inflammatory or ulcerative character. The effects are far more commonly local than general; and most cases of circumscribed chronic peritonitis owe their origin to some primary visceral lesion.

Certain regions of the abdomen are more likely to be affected than others. Of all these the pelvic peritoneum is the most frequently affected, and here the direct dependence of the inflammation upon the condition of the organs is most evident. Next in order of frequency are the hypochondria and epigastrium, where extensive changes of the diaphragmatic peritoneum may be traced to a pleurisy or pericarditis; a consequence to be explained by the close proximity of these structures, and by the direct lymphatic connection between the several serous cavities. Passing to the under surface of the liver, the peritonitis may be due to an ulceration (not necessarily with perforation) of the stomach, duodenum, gall-bladder or main bile-ducts, and new growths in this situation are a frequent cause of the same. Similar lesions of the cæcum and appendix, sigmoid and other flexures of the colon, and more rarely of other parts of the intestinal tract, as well as old hernias, may be the determining cause in their respective localities; whilst Baginsky considers that an intestinal catarrh may produce similar results.

Among the causes of certain obscure partial peritoneal inflammations most marked in the neighbourhood of the colic flexures, Virchow (1) has described fæcal accumulations which act as mechanical irritants to the bowel, and may become intensified in their effects by pressure, by a blow, or by intense peristaltic action.

Another very interesting form of peritoneal inflammation, chronic in course and more or less determined by the condition of certain of the viscera, requires special mention. Dr. Hale White (19) has given good reasons for regarding the majority of cases of so-called perihepatitis, both partial and complete, as merely a variety of chronic peritonitis, and usually accompanied with a splenic capsulitis which is of a similar nature. To what extent the liver and spleen immediately determine the peritoneal affection remains doubtful; but it is almost invariably associated with a granular kidney, and frequently with syphilis, gout, alcoholism, or obstructive heart disease.

(*e*) *Traumatic.*—Among the injuries which may give rise to a chronic peritonitis, repeated paracentesis is well recognised. Frequently restricted in its effect to the immediate neighbourhood of the puncture, it sometimes determines a general affection, possibly from a lack of strict antiseptic precautions. In very rare cases the symptoms date from an injury, such as a kick or a severe squeeze, which may have happened sometime

previously. Henoch records such a case in a child which prov
How far a lesion of some viscus, rather than a direct injury to
toneum, may have been the actual starting-point of the inflamma
generally remain doubtful, and may not be cleared up by post
examination. Long-continued pressure, as from tight-lacing, wou
capable of inducing the features of a slow peritonitis, accompanied
be, by few symptoms during life. Such accidents as the ru
abscesses, splenic infarcts, or cysts into the abdominal cavi
already been mentioned among the occasional causes of acu
tonitis; very seldom are they attended by symptoms which run a
course throughout. It may be observed that, whatsoever the n
the injury, the effects on the peritoneum are more likely to be l
circumscribed than general a l diffuse.

(*f*) *Associated with new growths.*—The relationship of perito
flammation to the development of new growths will be treat
conveniently in subsequent sections.

Among the various structural changes determined by the s
virus is a widespread fibrous hyperplasia; and as a part of this
the peritoneum may be extensively affected, especially in infa
children the subjects of the congenital disease. Prenatal perit
a chronic character is probably always due to syphilis; but it
to find certain visceral changes, such as cirrhosis, gumma,
lardaceous disease, coexisting with the peritoneal lesion (20).

The influence of sex and age.—Even after excluding pelvic pe
a greater number of subjects of a chronic non-tubercular peritoni
all causes are females; and, though no age is exempt, it is
more frequent in the first half of life. The evidence of the
among new-born infants has been already treated of, and the pr
of an acute idiopathic affection in childhood has been describe
such liability at this second age period obtains in regard to
peritonitis apart from tubercle; indeed, of twenty-four cases
by Tapret not one occurred before ten years of age. A consider
the causes just detailed would justify this expectation, for no
stances peculiar to this age are known to exert any special influer

Morbid Anatomy.—The post-mortem appearances in cases of
peritonitis exhibit considerable diversity, both in extent and in ch
The entire peritoneum may be involved, or the changes may be
limited to one or more regions, the rest of the membrane prese
normal appearance. Although there is this very obvious dis
between general and local peritonitis,—a distinction of cons
symptomatic importance,—the anatomical changes are for th
part identical in the two states, and differ only in extent and degr
a marked case of general peritonitis no part of the structure
exempt from the inflammatory changes; and, similarly, when the
are circumscribed there is scarcely any part which may not be
of them. Without doubt, however, the chronic manifestations of l
peritonitis—other than pelvic—are more commonly restricted

neighbourhood of the liver, the spleen, of the cæcum and appendix, and of the pylorus; in these situations, also, the affection gives the most marked evidences of its existence, and hence is most interesting clinically. But a peritonitis circumscribed at first may not always remain so; from it the inflammatory process may slowly extend until the whole of the serous membrane may become affected; or its progress may be marked by acute attacks in which, if death occur, both acute and chronic changes will be seen side by side.

A description of the post-mortem appearances met with in the various forms of chronic peritonitis falls most conveniently under the following heads:—(*a*) The Peritoneum itself; (*b*) the Adhesions; (*c*) Capsulitis; (*d*) the Effusion.

(*a*) *Peritoneum.*—The essential changes in the serous membrane itself consist in varying degrees of thickening due to the formation of newly-developed fibrous tissue, which is characterised by a great tendency to contract. From this cause the great omentum is frequently much increased in bulk, and, unless fixed by adhesions to the lower part of the cavity, is retracted towards the upper part of the abdomen, rather oftener towards the left side, forming a cord-like structure which occasionally appears to be subdivided into several irregular masses. The mesenteries are shortened, whereby the intestines are thrown into sharp curves and loops, and dragged backwards towards the spine, giving rise to the so-called "peritonitis deformans." The membrane itself loses its normal shiny, transparent appearance, and is dull and opaque; but a mere milky opacity, with no lack of gloss or lustre, is not to be taken as evidence of inflammation, for such a change is apt to ensue after death if the membrane become infiltrated with fluid. On its surface small spots may be seen resembling tubercle to the naked eye. Microscopical examination, however, shows that such is not their nature: nevertheless it is possible that such cases, treated by operation, may have been assumed to be tuberculous. Dr. Payne has described similar appearances in a case of cancer. The chronic inflammatory process is also marked by a very distinct pigmentation of the membrane and of the adhesions presently to be described. The colour may vary from a faint grayness to absolute blackness, and is distributed irregularly in streaks and patches over the entire peritoneal surface, parietal and visceral. That chronic venous congestion should increase pigmentation is illustrated under like conditions in the lung, in ulcers of the skin and otherwise.

The changes in the mesenteries and in the peritoneal covering of the intestines produce, in long-standing cases, a remarkable shortening in the actual length of the bowel, as well as a contraction in its diameter—all of which changes considerably interfere with the normal peristalsis.

(*b*) *Adhesions.*—The inflammatory process, besides producing a thickening of the serous membrane, usually develops new material in places where none should exist. Whether as a part of an acute attack, or in the course of the slowly-developing chronic changes, the inflammatory products, as regards their solid constituents, primarily consist of (i.) a

fibrinous clot, containing leucocytes and perhaps a few red blood-corpuscles, derived from the plasmatic exudation, which is an essential part of the inflammation. Into this fibrinous meshwork (the leucocytes and blood cells for the most part perishing and breaking up) grow (ii.) processes of large nucleated cells, the descendants of the proliferating connective tissue corpuscles of the fibrous framework of the normal peritoneum, and of those endothelial plates which cover the surface of the membrane and have neither disintegrated nor desquamated. The new cell elements from these two sources permeate the clot and, gradually giving rise to fibrous tissue, thus replace by permanent material the temporary adhesions which the plastic lymph at first maintained between opposed surfaces of peritoneum within the inflamed area. The "organisation" of the inflammatory product is further effected and established by the growth into these adhering bands of extensions from the blood-vessels of the normal membrane—the adhesions thus becoming constituent tissues of the affected part. The extent to which such adhesions may be found offers the greatest variety; from a few fine threads or membranous films easily broken down on handling, and occurring only here and there, they may extend to so extensive a formation of cords and bands as to weld the entire abdominal contents together into a mass from which the single viscera cannot be separated. Or this extreme condition may be limited to certain regions: thus the liver and the diaphragm may be so firmly bound together as not to be parted, even with the knife; or the pyloric region of the stomach may be closely attached to the under surface of the liver and head of the pancreas; or the spleen may be embedded in dense fibrous tissue which binds the organ to stomach, liver, and abdominal wall in an inextricable union; or again the cæcum and its appendix may be involved in a mass of similar material.

It sometimes happens that the adhesions may be sufficiently complete in one area to restrict the inflammatory process to that area, and thus a general peritonitis may be avoided; to this extent adhesions may be regarded as beneficial in their effects. But far oftener they are a source of trouble and even of grave danger. Tending as they do, after the fashion of scar-tissue generally, to shrink and contract, they may cause kinkings and constrictions of the intestine; or knuckles of the bowel may become nipped under fibrous bands, causing acute obstruction. The more general and extensive the adhesions, however, the less likely is strangulation to occur; nevertheless sometimes, owing to their situation and the traction exerted on them by the structures to which they are attached, the newly-formed bonds may yield and stretch. Occasionally they lead to considerable displacement of the abdominal organs.

(*c*) *Capsulitis.*—Occurring as a part of a general chronic peritonitis are certain special features connected with the liver and spleen. These organs may be covered with a white tough jacket of partially organised lymph, which may easily be peeled off, leaving the subjacent peritoneum intact, if deficient in smoothness and lustre, and the liver substance of normal character. Dr. Hale White, who has drawn attention to the con-

dition (19), notes the almost invariable association of it with granular kidney; and often with syphilis, gout, alcohol, and obstructive heart and lung disease. Included under the general names perihepatitis or perisplenitis, it differs from the true thickening of the capsule of these viscera, which is frequently seen, especially in the spleen; patches of this organ may attain a hardness almost cartilaginous, being formed of layers of tissue which have been compared to the corneal structure, and are like it non-vascular, while the substance of the viscus exhibits some degree of cirrhosis. Such appearances may be the only evidences of chronic peritoneal change; they may occur without any adhesions elsewhere or any effusion, and are probably set up by some such change as chronic congestion in the organ covered (7), rather than a primary affection of the serous membrane.

A very similar condition is frequently noticed in connection with granular contracted kidney, when the thickened capsule is closely fixed by old inflammatory material to the adipose and connective tissue in which it is embedded.

Spots of thickening due to the friction induced by tight lacing, and resembling those seen in the pericardium, are of common occurrence anywhere on surface of the liver and spleen; but the morbid change shows no disposition to spread.

The effect upon the underlying viscera of a thickening and contraction of their serous covering is not unimportant. Frequently the primary cause of the peritonitis affects the parenchyma of the organ or organs progressively with the peritoneum; or the inflammation may extend from them to the serous membrane: when, however, the latter is primarily affected the viscera may suffer secondarily, and for the most part the nature of the change consists in the development of an interstitial cirrhosis by extension from the peritoneum, with atrophy or degeneration of the proper tissue of the organ. A corresponding effect on the lung is known to follow a chronic pleurisy.

(*d*) *The effusion* in chronic peritonitis exhibits the greatest diversity in appearance and in quantity. Sometimes entirely wanting, at other times it may amount to many pints; but it rarely reaches such a quantity or causes such a degree of abdominal distension as occurs in simple ascites. When the viscera are so matted together as to obliterate the peritoneal cavity entirely its complete absence is intelligible, but the quantity when present bears no very close relation to the extent of the adhesions nor to the duration of the disease, whilst it certainly tends to vary in amount from time to time independently of treatment. How far the fluid is free in the abdomen will largely depend on the amount of adhesion existing between the coils of intestine, or between them and the other viscera and parietes; this may be so slight as to offer no resistance to the ready movement of the fluid, or it may confine the effusion to certain regions of the cavity from which it is dislodged with difficulty or not at all. Small collections of fluid may be completely encysted by the completeness of the limiting structures, and they appear to be less likely to be absorbed than when the liquid is quite free; the

density of the walls of these pseudo-cysts and their very restricted vascularity doubtless explain this retention.

In nature and appearance the exudation offers great variety. Premising that it is essentially an inflammatory product, and not a transudation of a serous fluid which has escaped from the vessels as the result of an increased pressure in the capillaries due to venous obstruction, it exhibits to a greater or less degree the power of coagulability; and, as such, approximates in nature to blood plasma. In some cases this power is but very slight, and only the merest threads of fibrin are seen; in others large flakes and clots are met with in the body after death, or they quickly form in the fluid which may be drawn off during life. At one extreme we meet with a clear straw-coloured or citron-tinted fluid, at the other extreme with pus; and between them appear all intervening degrees, described by such a term as sero-purulent, and dependent on the amount of leucocytes. Owing to the extreme delicacy of the new-formed vessels which develop in the adhesions they occasionally burst; hence blood in variable quantity may become mixed with the effusion. Cases have also been recorded in which, after repeated tappings, hæmorrhage has taken place between the layers of the newly-formed inflammatory tissue, more particularly on the abdominal parietes, forming numerous hæmatomas of varying size. Sometimes the effused fluid may be reddish brown or of a greenish tint from the presence of biliary pigments. In long-standing cases, or where the effusion is mainly purulent, curdy, cheesy masses are found in the cavity.

The various forms of chronic peritonitis, characterised by the different causal conditions by which they are determined, tend to be associated with certain of the morbid states here described as occurring in the disease as a whole, rather than with others. Thus, in the forms grouped under the general term "idiopathic," the effusion is oftener clear; unless it have followed a specific fever, when it becomes sero-purulent, the membrane is thickened and the adhesions are fewer. A very similar condition obtains in the cases which follow prolonged congestion of the abdominal viscera. In the chronic peritonitis resulting from toxic states, from injury, and from extension of inflammation from the viscera, the adhesions are far more numerous; and in the first of these conditions the effusion is more distinctly purulent. The liability to sanguineous effusion has been noticed especially when the peritoneal inflammation is associated with alcoholic cirrhosis of the liver. The peritonitis due to syphilis is singularly free from effusion, and the most characteristic and extreme degrees of "peritonitis fibrosa" (non-tubercular) come within this category; though, on the other hand, the change may be represented by a few bands and adhesions only.

Peritonitis, when strictly circumscribed, is generally chronic, and is almost always determined by some morbid condition of immediately adjacent structure. The encysted effusion may be clear, turbid, or purulent; and sometimes, owing to breaking down of the retaining adhesions and escape of fluid into the peritoneal cavity, a general peritonitis more

or less acute in character may ensue. When purulent it has been known to burrow in various directions, sometimes to great distances behind the peritoneum, and even to burst into the intestinal tract or into the pleural cavity. The encysted collections of fluid, by the pressure which they exert, may cause displacement or even partial atrophy of viscera.

Signs and Symptoms.—The signs and symptoms of chronic peritonitis, whatever its cause, will in any individual case be largely conditioned by the relative proportion of the effusion and of the adhesions. Hence arises considerable variety in extent of manifestation, and in many cases such obscurity of evidence that the diagnosis may long be held in doubt, and finally reached by exclusion only. Even when the signs of abdominal mischief are tolerably evident the diagnosis is made rather by a consideration of them, and of the history of the case collectively, than by the presence of any one crucial symptom. Whilst in one set of cases the physical signs may be fairly obvious, in others they may be scarcely appreciable; the symptoms that occur with marked prominence in one patient may be wholly or almost wanting in another; and the exclusion of tuberculosis will always be a difficulty, often a difficulty left unsolved.

The *physical signs* of the malady are usually present to some degree and are often well marked, though they are liable to vary as the case progresses. The degree of emaciation of the patient largely depends on the duration of the disease, the extent of the digestive disturbance, and the nature of the inflammatory process—being greatest as a rule when the fluid tends to be purulent. The skin is generally harsh and dry, often it is particularly so over the abdomen. When effusion is but slight or wanting the abdomen is sometimes flattened and retracted; but, as fluid accumulates, it presents all degrees of distension especially if there be any meteorism, which is frequent enough. The distension, unless very excessive, is rarely quite uniform, or even constant in irregularity; there may be rather more bulging over the gastric area from a retracted thickened omentum, or the most prominent region may be in one of the flanks; moreover, as the flatulence varies or the fluid shifts, the contour of the abdomen will change likewise. Irregularities of shape may be equally noticeable when the belly is retracted; and the movements of the intestines are frequently visible. Probably the most valuable evidence is that obtained by palpation. A general sense of increased and often irregularly-distributed resistance is communicated to the hand of the observer; and at places a feeling of very distinct hardness, as of nodular but slightly movable lumps; with this there is often a characteristic and quite peculiar sensation, imperfectly described by the word "doughy"; this is caused by the juxtaposition of distended and semi-distended coils of intestine, and firm, resistant tracts of new-formed inflammatory tissue and small collections of liquid. When the effusion is considerable, and not encysted, fluctuation and fluid may be perceived; yet in this latter case the evidence of fluid may be very indistinct.

"Under certain conditions," says Dr. F. T. Roberts, "the abdomen yields a peculiar feeling of being movable as a whole." It is occasionally possible to demonstrate the existence of adhesions between the viscera and abdominal walls by compressing the surface a short distance from the suspected region, thereby causing the integuments to become raised up over the site of the peritoneal attachments. In rare cases a friction fremitus may be detected. On percussion, if the peritoneal cavity be greatly distended with fluid, a uniform dull note is obtained; if not, irregular areas of resonance and dulness are to be recognised which correspond to the stomach, the intestines, and the inflammatory products, both solid and liquid; as the liquid shifts with the movements and manipulation of the bowels the dull regions are constantly changing, and it is common to find a resonant flank and a dull flank presently to change places, though, unless the fluid be large and the adhesions few or none, such alterations may be but little marked on altering the patient's position.

Even less uniform than the physical signs are the *symptoms* complained of by the patient. At first they are vague and uncertain, coming on gradually and insidiously, and seldom in such a manner as to indicate even the seat of the malady. When supervening on an acute attack the history of the case may suggest the real nature of the disease, but otherwise it is likely to remain doubtful for a long time. Ill-defined sensations of a dragging character, or other discomforts, now and then amounting to actual pain and soreness, may be the first symptoms which are noticed. Even as the disease progresses the evidence of pain is most variable; at times and for long periods it may be almost or quite absent, at others distressing and severe; possibly it varies as fresh tracts of the peritoneum become involved or acute exacerbations of the malady occur. It seems pretty certain that the inflammation of a previously healthy serous membrane gives rise to more severe pain than when the structure is already in a condition of disease. Pain, when present, is often aggravated by movement, especially by walking downstairs; and relief is often felt by the firm pressure of a bandage or other support. The abdomen is generally tender to the touch, at any rate in places, and at times this may even render contact with the bed-clothes unbearable; but no certainty or regularity can be affirmed of this symptom. The temperature may remain normal for weeks, or even subnormal, now and then interrupted by irregular attacks of pyrexia when the thermometer may register as high as 103° F. Sooner or later the bodily strength is impaired; and a slowly-progressive weakness is notable with disinclination to exertion. The pulse is small and quick, and respiration is apt to be more frequent than normal from the impediment offered by the state of the abdomen. The greater number of the symptoms, as might be expected, are concerned with the digestive function and the alimentary canal. The appetite as a rule is poor, though sometimes it is scarcely affected; but the taking of food frequently determines attacks of colic, which are due to spasmodic contraction of the bowels, hampered as they are in the rhythm of their peristalsis by the adhesions. In this way collections of

flatus, the result of arrested digestion, become locked up in sections of the canal, giving rise to pain and discomfort until released. All degrees of constipation, often amounting to such complete obstruction as to necessitate operative interference, are produced by the same cause; the shortened bowel may be kinked or twisted, the muscular coat atrophied by the pressure of the thickened peritoneal covering, and bound down here and there by fibrous bands; beneath one of these a knuckle of the tube may become acutely constricted, and indeed in every way impediment may be offered to the normal action of the intestine. Vomiting and diarrhœa are very occasional in their occurrence.

The presence of bands of fibrous tissues, the adhesions of the viscera to one another and to the parietes, lead to very varied pressure effects; thus œdema of the lower extremities, thrombosis, or neuralgic pains, may result from pressure on the large vessels and nerves in the abdomen; or jaundice may follow from compression of the main bile-duct; or micturition may be rendered difficult and painful, or the adhesions may be responsible for any intractable dysmenorrhœa. To remember that abdominal pains, often localised, and in their manifestation simulating gastralgia, gastric ulcers, hepatic or even renal colic, may depend upon old adhesions, is of great practical importance; especially as such cases are far from uncommon, and not infrequently amenable to treatment. The diagnosis of such adhesions, however, is generally difficult, and often to be arrived at by exclusion only. There may be little or no history available to suggest the effects of a former peritonitis, and the patient's story may be such as to indicate almost certainly the presence of one or other of the morbid states mentioned above; some one symptom may, however, be wanting to complete the diagnosis, and then, unless the possibility or even probability of old adhesions be remembered, the case may go unrecognised and unrelieved. The abnormal adherence of viscera to one another and to the wall of the abdomen might not unreasonably be supposed to interfere with the blood-supply of the organs; and in this way uterine adhesions within the pelvis may explain some cases of menorrhagia.

The progress of chronic peritonitis is rarely if ever towards recovery. The symptoms may abate, but the anatomical conditions for the most part remain, to be a constant source of fresh outbursts of subacute or even acute character. When the clinical indications are at all well marked the course of the disease is almost always to an unfavourable termination, though this may not be reached for several months or even for years. The causes of death are usually wasting and exhaustion, aggravated in neglected cases by bed-sores, or by a chronic suppuration discharging possibly into the bowel, or elsewhere.

Diagnosis.—From the clinical features described, their great range of variability, and oftentimes uncertain character, the diagnosis, as I have already said, is frequently difficult; the exclusion of the presence of tubercle is doubtful at all times, and often impossible. The evidences of the condition which are probably the most conclusive are the peculiar appearance and feel of the abdomen. If the peritoneal cavity be fully

distended with fluid even these characteristics may be wanting, and a precise diagnosis of the real nature of the state must necessarily remain in abeyance. A consideration of the causal conditions which I have fully described shows that the history of the case may sometimes assist in the diagnosis, at least so far as to suggest the probability of peritonitis. Even in the most experienced hands mistakes are not unlikely, especially in confounding the lumps caused by the inflammatory thickening with masses of new growth of sarcomatous or carcinomatous character: the abdomen has now and then been opened in the full expectation of finding a neoplasm, but only to discover an extreme condition of peritonitis fibrosa.

Prognosis.—No very general statements can be made regarding the prognosis of the cases here described. At the best, intervals of relief may be hoped for but scarcely promised, and complete spontaneous cure, when the symptoms are at all pronounced, is not to be expected. The greater gravity of those cases in which suppuration continues has been already referred to; and in marked contrast to these are the cases—not a few in number—in which the anatomical evidences of the disease, of which scarce any symptoms were manifested during life, are revealed only on post-mortem examination. The injurious mechanical effects of the adhesions and bands are but ill compensated, as a rule; and their results are therefore lasting, and constitute an ever-present risk to life by causing intestinal obstruction: whilst the less acute phenomena, such as pain, chronic suppuration, and digestive failures, tend to serious impairment of the general health. One point in regard to the prognosis of those cases of chronic peritonitis described by Dr. Hale White which are characterised anatomically by perihepatitis, and clinically by the presence of ascites, is the more favourable course they run after tapping than do those in which the dropsy is due to alcoholic cirrhosis without any peritonitis. The latter patients, according to this author, hardly ever live sufficiently long to permit a second operation, whilst should life be so prolonged as to admit of it "the diagnosis of cirrhosis of the liver is almost certainly either completely wrong, or else the patient has something else the matter with him in addition to cirrhosis; and, further, it is highly probable that he has some form of chronic peritonitis, of which what is ordinarily called perihepatitis is merely a variety" (21). [*Vide* art. "Perihepatitis," vol. iv.].

Treatment.—The main indications are to remove the inflammatory products as far as possible, and to maintain the general health of the patient. The former object can seldom be attained completely. Extensive fluid effusions should be removed by paracentesis; and the operation should be repeated without hesitation as often as necessary. When the exudation is simply serous permanent relief may be looked for after one or two tappings; but such a result rarely follows if the effusion be at all purulent. Efforts may be made to obtain absorption of the products by mercurial or other applications to the abdomen; of these inunction with unguentum or oleatum hydrargyri, or linimentum hydrargyri

sprinkled on lint, and painting with tincture of iodine, are the most usually employed. These measures, however, can seldom be used for a long time, as they are liable to blister or to produce the effects of mercurial poisoning; and in my opinion more benefit is to be obtained from the free application of a dilute solution of tincture of iodine (one in three parts of water) night and morning, an application which, as a rule, can be tolerated for a considerable period.

The pain and sensations of discomfort, when acute, frequently call for treatment; hot fomentations, either sprinkled with tincture of opium, or applied to the abdomen which has been thickly smeared with a pigment composed of equal parts of extract of belladonna and glycerine, usually give relief. Small doses of opium or morphia internally may be necessary to subdue the pain which tends, unless checked, to continue, and thereby to interfere with the sleep, the appetite, and the general well-being of the patient. As a rule pressure is well borne, and much benefit and comfort follows the wearing of a tightly-adjusted belt or coil of flannel; the vague but distressing symptoms comprised within the word "sinking" are much relieved thereby, probably by counteracting the dragging exerted by the peritoneal adhesions, or it may be by emptying the mesenteric venous reservoirs. The impunity with which, under proper precautions, the abdominal cavity may be opened, warrants the performance of laparotomy when the pain is persistent and intolerable, chiefly with a view to a separation of adhesions. Even if these have proved too extensive for complete division, very distinct relief has been known to follow the mere opening of the abdomen, whilst the risks of such a procedure are now reduced to a minimum. How far the operation may determine the permanent benefit which, as will be presently seen, frequently follows it in cases of tuberculous peritonitis remains uncertain; we have not records sufficient to determine the matter, but at first sight it is not likely that such satisfactory results would follow this treatment in non-tuberculous cases as in those where the affection is primarily peritoneal; the remote causes of the cases now under consideration are scarcely to be influenced by the simple opening of the abdomen.

One of the most troublesome symptoms is the constipation, which may become extreme. Violent purgatives are certainly to be avoided; as a rule the milder vegetable cathartics, such as senna, cascara, and the like, appear to act more satisfactorily than the saline aperients, and from time to time these drugs may be supplemented by enemas of castor oil.

The general health should be sustained, as best it may, by such food as may combine the most nutriment with the least indigestible residue; hence milk, eggs, cream, cereal foods, soups, and small quantities of meat, should form the staple diet. Syrup of the phosphate or iodide of iron, and Easton's syrup, are among the tonics which may be given with most advantage, together with maltine and cod-liver oil.

W. H. ALLCHIN.

REFERENCES

1. BAUER. Ziemssen's *Cyclopædia of the Practice of Medicine*, English trans. vol. viii. p. 292, 1878.—2. CROOKE, G. F. *Path. Soc. Trans.* xxxix. 1888.—3. FAGGE. *Principles and Practice of Medicine.*—4. *Idem. Brit. Med. Jour.* 1876, i. p. 71.—5. FLINT, AUSTIN. *Brit. Med. Jour.* 1883, ii. p. 567.—6. HABERSHON. *Diseases of the Abdomen*, 4th edit. 1888, p. 651.—7. *Idem. Trans. Path. Soc.* 1876, vol. xxvii. p. 79.—8. HENOCH. *Deutsch. med. Woch.* 1892, Jan. 7th.—9. HERRINGHAM. "Peritonitis in Bright's Disease," *St. Bartholomew's Hosp. Reports*, 1893.—10. JOHNSON, Sir G. *Brit. Med. Jour.* 1876, vol. ii. pp. 243, 359.—11. LEUDET. *Clinique de Rouen*, 1874.—12. MEAD, R. H. *Brit. Med. Jour.* 1876, vol. ii. p. 393.—13. OSLER. *Pract. of Med.* 1895, p. 506.—14. PAYNE. Discussion on the Pathology and Morbid Anatomy of Chronic Alcoholism, *Path. Soc. of London*, 1889.—15. PYE-SMITH, P. H. *Path. Soc. Trans.* 1882, vol. xxxiii. p. 172.—16. SPILLMAN and GANZINOTTY. *Dict. encyc. des sciences médicales.*—17. TAPRET. *Thèse de Paris*, 1878.—18. VIERORDT. *Thèse de Tubingen*, 1884.—19. WHITE, HALE. *Clin. Soc. Trans.* 1888, vol. xxi. p. 219.—20. *Idem. Trans. Path. Soc.* 1888, vol. xxxix.—21. *Idem. Guy's Hospital Reports*, vol. xlix. 1892.

W. H. A.

TUBERCLE OF THE PERITONEUM

(TUBERCULOUS PERITONITIS)

THE specific form of new growth called "tubercle," which is determined by the invasion of the peritoneum by the tubercle bacillus, may and usually does give rise secondarily to an inflammation of the serous membrane—tuberculous peritonitis. The varieties in clinical manifestation and in post-mortem appearances exhibited by the cases comprised under this name are due mainly to the different degrees and extent of the inflammatory process, and to the proportions in which the various inflammatory products are formed. At the one extreme the affection consists of gray granulations only, or is associated with so little inflammation that the latter is scarcely obvious; at the other the true tuberculous element is so surrounded and embedded in the structural results of its own changes and of the accompanying inflammation that it can hardly be found.

It will be more convenient to describe the anatomical conditions met with in the disease before discussing the manner in which it originates, and its relation to tuberculosis of other regions. Except so far as concerns the etiology of this condition, much that has been said in the previous section in respect to the morbid anatomy and symptoms of chronic peritonitis is applicable likewise to the present form of peritoneal inflammation.

Morbid anatomy.—The simplest appearance is that presented by *miliary tubercle of the peritoneum.* Scattered over the membrane are numerous small pearly translucent nodules, from the finest perceptible up to

the size of a pin's head; usually discrete, with here and there several confluent—the well-known "gray granulations." There may be but very few, the entire serosa may be so covered with them as to give a distinctly granular feel to the touch, or they may be limited to either the parietal or visceral layer, and in the latter situation may be restricted to areas corresponding to tuberculous ulcers of the mucous membrane whence they have spread, by direct extension, to the hepatic or splenic coverings, or to the surface of the pelvic viscera; or again, if wide-spread, they may be most abundant over the diaphragm, the mesenteries and omentum. Further examined, it will be seen that each tubercle is frequently situated in close connection with a small blood-vessel or lymphatic, upon the wall of which it may be said to develop itself; in other cases it lies on the serous membrane itself in the meshes of the vascular network (see article "Tuberculosis," vol. ii. p. 3).

This process being essentially acute or subacute in its course, caseation is seldom to be perceived, and there is either no coincident inflammation, or at the most it is represented by a small zone of hyperæmia surrounding each granulation, with points of hæmorrhage, and perhaps a slight dulness of the normal shining surface of the peritoneum with some desquamation of the endothelium and "stickiness" to touch due to the merest film of fibrinous exudation, sufficient at spots to cause the slightest adhesion of adjacent layers. When the tubercles have been of long duration there may be a deposit of pigment around them, together with a delicate translucent material, the earliest stage of the fibroid change. In many cases there is an effusion of straw-coloured or sanguineous fluid, often amounting to many pints, and differing from the serum of simple ascites due to portal obstruction by being more or less coagulable; it often forms a firm jelly after withdrawal. Rarely is the exudation sero-purulent in character. The mesenteric glands are enlarged and the seat of tuberculous invasion.

Such are the appearances when limited to the specific tuberculous elements of the disease, but just as these "gray granulations" are themselves the structural response to the irritant represented by the tubercle bacillus, so in turn the granulations set up inflammatory changes of varying extent in the surrounding membrane. Certain alterations, moreover, occur normally in the tubercles themselves, and contribute to the anatomical appearances which characterise the disease. The most important of the changes originating from the gray granulations and the associated inflammation is a development of fibrous tissue; another consists in the caseous degeneration of the new growth, and to some extent of the inflammatory products; and, lastly, an effusion of fluid is a common feature of the inflammation. The degrees to which these various morbid manifestations are present constitute the anatomical differences met with in the disease, and mainly determine the clinical varieties. The duration of the malady is obviously an important factor in determining the extent to which these appearances are developed; for while in the acute cases, as already seen, little more than the tubercles are formed in those

which have lasted some time, the neoplasm is combined with the structural alterations, even to the extent of being almost obscured. But besides the formation of new tissue (*fibrosis*) and the degeneration of the tubercles (caseation), the perverted nutritive processes known as inflammation entail, to a greater or less extent, an actual destruction of substance by molecular necrosis (ulceration): and this condition is frequently met with in chronic tuberculous peritonitis, of which, indeed, it may be a prominent feature. According as the fibrous tissue formation or the ulceration predominate in the progress of the tuberculous peritonitis, may the disease be described as "fibrous" or "ulcerous": but no marked line of separation exists between these forms which are mutually connected by all intermediate degrees of one or the other condition. In an extreme case of *fibrous tuberculous peritonitis* the individual tubercles are scarcely to be found, embedded as they are in fibrous tissue to the formation of which they themselves contribute by their own conversion into that material as one of the natural directions in the evolution of the new growth. Tough, firm, membranous bands and adhesions are thus formed by which the abdominal viscera are matted together and bound to the parietes. The intestinal coils are shortened and contracted, and the mesenteries and parietal layer are considerably thickened and inextricably involved in the general mass. Such a condition is not infrequently the final stage of some cases of miliary tuberculosis of the peritoneum; the continued development of the bacillus being arrested, the fluid effusion is absorbed, and a general fibroid change slowly supervenes. Even the ulcerous form in favourable circumstances may occasionally terminate in this way. Where the extent of adhesion of the organs is not so extreme, the intestines are frequently drawn backwards towards the spine or towards the right upper region of the abdomen. This displacement is owing to the attachment of the mesentery to the lumbar vertebræ being normally directed obliquely downwards towards the right sacro-iliac synchondrosis; hence when the suspending fibres are shortened the intestinal loops are dragged towards the right. The similarity of the condition presented by this extensive fibrosis to that induced by the syphilitic virus is noteworthy.

In the ordinary *ulcerous form* the tubercles exhibit a greater degree of caseation—the other direction in the normal evolution of the new growth—forming masses of cheesy matter up to the size of a marble, together with fibrous bands, and gray granulations recently developed and in all stages of degeneration; and of no less importance are enlarged and caseous mesenteric glands. In this way very considerable masses of material are formed which exhibit various degrees of necrosis and breaking down, involving in the ulceration the tissues of the organs with which they are in contact, and which they serve to bind together. Between contiguous coils of intestine, or between them and the abdominal wall, ulcerating areas of this kind may be seen, often establishing communication between adjacent knuckles of bowel; the adhesions forming more or less complete loculi, in which are contained encysted collections of fluid of a sero-fibrinous or sero-purulent character, or even actual abscess which

may be fæcal. As in other forms of chronic peritonitis there is a great tendency for the new-formed tissue to become pigmented, grayish, or almost black. Occasionally in the caseous masses small calcareous particles are to be met with. The intestinal walls become softened and easily tear in handling. It is in association with this caseous ulcerating variety that the omentum is likely to form large irregular cords, being infiltrated in tracts by cheesy tubercle, inflammatory new growth, and even encysted collections of exudation. These omental tumours are usually situated in the upper region of the abdomen along the course of the transverse colon, often more marked towards the left side; but occasionally when the omentum is fixed by adhesions the tumours may be found elsewhere, as in the right iliac and lumbar regions; but the lumps formed as above described may occupy the iliac or hypogastric regions, or extend into the flanks, until the greater part of the cavity appears to be filled by them. Such masses as these are likely to cause œdema of the lower extremities, or even of the trunk, by pressure on the large veins, or by obstruction of their channel by tuberculous invasion of the vessel-walls and consequent thrombosis.

It is in this class of cases that a very characteristic manifestation of the disease is frequently met with; namely, a development of tubercle, with caseation and ulceration, in the connective tissue around the fœtal remains (urachus, obliterated hypogastric vessels, etc.) connected with the umbilicus, at which a discharge frequently takes place through a sinus leading from the surface to the necrotic area; and as this again may communicate with the bowel as above described, the mischief may lead to the formation of an umbilical fæcal fistula. The integuments around the navel are red and œdematous.

In a small proportion of cases the enlargement and caseous degeneration of the mesenteric and retroperitoneal glands may be so considerable as to form large nodular tumours and constitute the predominant manifestation of tuberculosis; the peritoneal affection being quite subsidiary.

An effusion in chronic tuberculous peritonitis is often wanting; indeed, perhaps, it would be more correct to say that this form of peritoneal inflammation is usually "dry." Some amount of exudation, however, is not infrequent, and is generally turbid or blood-stained, or even purulent; when this is so, the pyogenetic infection has gained entrance to the serous cavity through the intestinal walls which are the seat of tuberculous ulceration. In this tendency to be associated with pus-formation tubercle differs markedly from syphilis, although in respect to fibroid formation these two chronic infective diseases resemble each other. It is not often that the tubercle bacilli are discoverable in the effusion, even when the tuberculous nature of the disease is undoubted. There is reason to believe that the germs chiefly remain and propagate on the surface of the serous membrane, and are not shed to any great extent into the fluid.

The frequent association of chronic tuberculous peritonitis with cirrhosis of the liver has long been noticed. The serous inflammation is

secondary in point of time, and is of the fibrous rather than the ulcerous variety; but owing to the portal obstruction produced by the change in the liver there is a coexistent ascites, and E. Wagner has described a curious brownish-yellow colouring of the external surface of the small intestine. The coexistence is far more frequent in men and in alcoholics. Coexistent tubercle in the spleen is frequently found, and the capsule of the organ is particularly liable to be thickened with tuberculous growth.

A tuberculous peritonitis is very rarely restricted to a small area, though the disease may sometimes be strictly confined to the situation of tuberculous ulceration of the intestines, adhesions binding adjacent parts together and limiting the distribution of the affection; a similar restriction may be found in connection with tubercle as it affects the pelvic viscera.

Pathology.—Immediately determined as this disease is by a specific organism—the bacillus tuberculosis—we must now inquire by what channels the virus gains access to the peritoneal cavity.

Tubercle, it must be remembered, is a local affection which naturally tends to undergo either a fibrous or caseous transformation. In the latter condition it may break down, the bacillus-containing débris being thereby liable to become diffused throughout the body by the vascular or lymph channels, or by direct invasion. By each of these routes may the peritoneum be infected.

The extensive distribution of the gray granulations and their close relation to the vessels at once suggest the blood current as the course by which in acute miliary tuberculosis the germs reach the serous cavity, whatever may be the exact way they originally gained entrance to the blood stream. It is as a part, therefore, of a general tuberculosis that the peritoneal affection may be met with, although the membrane is less often involved than other structures, and frequently escapes even in a widespread invasion.

A primary tuberculous peritonitis is of very doubtful existence, and the disease is probably never limited to the peritoneum, though it may be confined to the abdomen. Most commonly an eruption of tubercles on the abdominal serous membrane is secondary to pulmonary tuberculosis and caseating lymphatic glands in the thorax, the infection being conveyed, in all probability, by the lymphatic vessels. In a valuable communication on the distribution of tubercle in abdominal tuberculosis, based on sixty cases observed during one year at the Children's Hospital, Great Ormond Street, by Dr. W. S. Colman, nine cases, or 15 per cent, are mentioned as presenting gray granulations limited to the parietal layer of the peritoneum covering the diaphragm and anterior abdominal wall; in only two of these was there any tubercle in the intestines or mesenteric glands, whilst in every one there was advanced caseation of the thoracic lymphatic glands and pulmonary tuberculosis.

In the caseating ulcerous forms of peritonitis there is a much greater frequency of coexisting tuberculous disease of the intestines or

mesenteric glands, the chest-lesions being often wanting. Dr. Colman found seven cases (12 per cent) of this condition, in all of which the mesenteric glands were caseating; in four there was tuberculous ulceration of the intestine, but in only one was there any tubercle in the thorax. In such cases the path by which the virus reaches the peritoneum is most likely from the intestines by way of the lymphatic vessels and glands, often without any lesion of the mucous surface; or a direct invasion of the serous cavity, by the rupture into it of a caseating mesenteric or retroperitoneal gland, has been known to occur. Other sites of tuberculous disease from which the peritoneum may become infected are the vesiculæ seminales, the epididymis and testis, the Fallopian tubes, and more rarely the vertebræ or the coxo-femoral joint. Dr. Osler thinks "that in 30 to 40 per cent of the cases in women the tubes are found affected, usually as a primary lesion from which the peritoneum is infected, less often secondary to the peritoneal affection." As a possible but most exceptional mode by which the bacillus may gain entrance to the serous cavity traumatism may be mentioned.

In connection with the close association of tuberculous peritonitis in its ulcerous form with caseating mesenteric or retroperitoneal glands, it may be well to note the comparative frequency with which these organs are attacked in all cases of the disease. Dr. Sims Woodhead, of 127 cases of tuberculosis in children, found these glands involved in 100 (78·7 per cent), and "in 14 the mesenteric glands only were affected, that is, there was no tubercle found in any other part of the body. These 14 cases were accompanied by neither ulceration nor cicatrisation of the intestine, and there was no peritonitis"; the gland-lesion was essentially primary, a condition which can probably never be affirmed of the peritoneum. Dr. Colman found tuberculous mesenteric glands in 66 per cent of his cases, and Dr. Walter Carr, in a series of 120 cases of tuberculosis in children, noted the mesenteric glands were affected in 65 (54 per cent); Carr concludes from post-mortem examination, and having regard not so much to the proportionate frequency of caseation in particular glands as to the group in which the process is most advanced, that is, in which the disease began, "that tuberculous disease starts much more frequently in the thorax than in the abdomen, and certainly far more often in the thoracic than in the mesenteric glands." He also enters a useful caution against "being misled by the far too frequent use of that much-abused term 'consumption of the bowels,' which, as employed by the laity, indicates simply marasmus due to improper food and very rarely any tuberculous lesion"; on this point Professor Allbutt had already spoken in the same sense (6). The frequency with which the mesenteric glands may be found affected after death very considerably exceeds the occasions on which they may be detected as enlarged during life; and Dr. Frederick Taylor's opinion than "an amount of disease of the mesenteric glands which can be recognised clinically as such—tabes mesenterica—is uncommon as compared with recognisable tuberculous ulceration and recognisable peritonitis" (3), is

quite in accordance with general experience. As the chief and primary lesion, the glandular affection apart from peritonitis is clinically rare, whatever be its frequency as a pathological occurrence. Susceptibility of these glands is easily understood when their liability to irritation from even transient intestinal derangement is remembered. Occurring also at a period when their activity is at its maximum, and their liability to disturbance is presumably greatest, circumstances combine to diminish their normal resistance to microbic invasion, and to make them a place of lodgment and development for such tubercle bacilli as may reach them from the alimentary canal, in many cases apparently without perceptible lesion of the mucous membrane. Dr. Sidney Martin fed animals on tuberculous sputum, and found that the bacilli passed through the intestinal walls without any ulceration of them (*vide* also art. "Tuberculosis," vol. ii. p. 22). Dr. Colman's statistics (p. 658) tend also in the same direction. The germs may remain in the glands for a variable time, perhaps without further spread; or some circumstance may favour their dissemination from these glandular foci when the peritoneum, either alone or as a part of a more general infection, may become the seat of tuberculous disease.

When the peritoneal tuberculosis is mainly miliary in character and specially involves the parietal layer, one or both pleuræ may be affected, the disease spreading from one serous membrane to the other. In such cases the tubercle appears to be restricted to these structures and the glands, and not to invade the viscera; this has been explained by supposing that the inflammatory exudation, thrown out on the surface of the membrane, blocks up the stomata and thereby hinders the spread of the infection by the lymph channels. Pleurisy developed in this way is frequently "dry."

The general conclusions to be drawn from the foregoing observations would seem to be that tuberculous disease of the peritoneum is of frequent occurrence, whether as a part of a generalised tuberculosis or as constituting the predominant manifestation of the disease; that the peritoneum may become infected from the intestinal canal by direct extension from tuberculous ulceration of the mucosa, or, as appears probable, without any recognisable lesion of the intestine, the virus in such cases reaching the mesenteric glands, from which the serous membrane may become subsequently affected; further, that the disease both of the peritoneum and abdominal glands may be secondary to tubercle in the thorax, the channel of infection then being by lymphatics or blood-vessels. And a remarkable fact in respect to the association of tuberculous peritonitis with tuberculous disease elsewhere is the comparative rarity with which tuberculous ulceration of the intestine accompanies the peritoneal affection; and, whilst the intestinal lesion as a rule coexists with pulmonary tuberculosis, this latter condition seldom accompanies the caseating and ulcerous form of peritonitis, though it is often found with a miliary tuberculosis of the peritoneum. Finally, by whatever means and from whatever sources the serous membranes of the chest and

abdomen may become affected, the disease, though it may spread from one to the other, rarely does so from them to the viscera.

The means by which the organism finds admission to the intestinal canal, whether by food such as milk and meat, or by the swallowing of infected sputum from tuberculous lungs, is discussed elsewhere [see article "Tuberculosis," vol. ii.], as also the conditions which may diminish the resisting power of the intestinal mucosa and so favour the invasion of the microbe. Among the latter would appear to be an intestinal catarrh or a chronic congestion of the portal area, such as would be caused by cirrhosis of the liver; thus the association of the hepatic affection and tuberculous peritonitis already referred to may be explained, or both tubercle and cirrhosis may be favoured by alcoholism.

Such of the remoter causes as can be in any degree recognised are not known specially to affect the incidence of the disease on the peritoneum, but are involved in the general etiology of tuberculosis. Overcrowding and defective ventilation, rickets and the acute specific diseases, especially measles and whooping-cough, are probably the most effective of all such factors. It is very doubtful whether sex affects the occurrence of the disease, although post-mortem records show a preponderance among males, especially of the fibrous form; and as regards the influence of age, it is certainly very frequent between the ages of three, or four, and twenty years, when the tissues of the body approximate more nearly to the embryonic type, and may perhaps be presumed to be more responsive to morbid influences; though it is far from being restricted to that period of life, having been met with at an advanced age. On the other hand it is almost unknown under two years of age. Dr. Osler considers it is most common between the ages of twenty and forty. Hereditary predisposition is certainly not well marked.

Course, signs and symptoms.—The cases in which the abdominal serous membrane is found affected with tubercle fall into two great clinical groups, the acute and the chronic—a distinction which is for the most part well maintained, notwithstanding that the disease now and then runs a course that would justify its being placed as intermediary between these divisions, and also that many of the chronic cases are liable to manifest acute exacerbations from time to time. The signs and symptoms are, however, mainly due to the peritoneal inflammation rather than to the actual tubercle with which it is associated.

(*a*) *The acute form.*—From the foregoing account of the morbid anatomy and pathology of this affection, it is apparent that tubercle of the peritoneum, with or without any associated peritonitis, may occur as a part of a generalised tuberculosis; or it may constitute substantially the entire disease. Corresponding in great measure to this difference in anatomical distribution are the symptoms manifested, though not entirely so. Thus in cases of a general dissemination of tubercle, it often—probably usually—happens that symptoms specially referable to the abdomen are few or wanting, and only evidences of a general febrile disease are to be recognised. Likewise in many of those cases where post-mortem

examination shows the peritoneum to be the seat of an extensive growth of tubercle, indications of its presence during life may have been completely absent or were overshadowed by the symptoms due to tubercle in other organs.

The clinical features of acute general tuberculosis, the frequent absence of signs of local affection, the resemblance in many points to enteric fever, and its lapse into the "typhoid state," have been fully described elsewhere [see article "Tuberculosis"]; it remains here to refer to those characters which distinguish the acute disease, so far as may be, when the abdominal symptoms predominate. Speaking generally, such symptoms are proportionate to the extent of the associated peritonitis, which has been above shown not to be an invariable accompaniment of an eruption of miliary tubercle. By acute cases are meant those which run a course of one or two, rarely three months, and for the most part terminate in death; and among such the sole evidence of the abdomen being involved may be that derived from the presence of an effusion into the peritoneal cavity—the physical signs, that is, of a simple ascites unaccompanied by pain or any other local symptoms. In other cases, often when the effusion is not so great, pain and tenderness over the abdomen, not necessarily in any special region, nor yet constant in position, may constitute a marked feature, and may precede the indications of the dropsy. A flatulent distension of the intestines, amounting perhaps to a condition of tympanites, may be an early symptom, afterwards to be followed by the fluid effusion; this will of itself add largely to the patient's discomfort and intensify the pain, to which also a coexistent dry pleurisy may contribute. Either constipation or diarrhœa may be present, or these states may alternate; vomiting has been occasionally noticed. The general manifestations, such as malaise, headache, pyrexia of a hectic type (though with a very frequent liability for the maximum temperature of the twenty-four hours to occur in the early part of the day), loss of appetite, furred or dry tongue, thirst, hot dry skin, quick small rapid pulse, progressive emaciation, together with signs of tubercle elsewhere, are among the symptoms of general tuberculosis; though they vary considerably in the extent to which they may predominate over those which are referable to the abdomen, or be subsidiary to them. As a general rule the onset of symptoms is more or less gradual, though often without any history of previous ill health; and seldom are they so sudden as to suggest enteritis or internal strangulation, yet such cases have been recorded when an operation for the relief of the obstruction has revealed an unsuspected tuberculous peritonitis (10, 13, 17, 18). Death in such cases is rarely if ever attributable to the abdominal lesions, but rather to a progressively deepening typhoid condition ending in coma.

(*b*) *The chronic form.*—This division includes those cases, far more numerous than the former, which may last for many months; they tend, as a rule, to end in death, but they are frequently varied by periods in which the symptoms subside to a very considerable extent: on the other hand they may assume an acute character.

The marked difference in the structural changes which distinguish the (i.) ulcerous caseating and (ii.) fibrous forms respectively, determine to a great degree the differences in physical signs and symptoms which are met with in these two classes of cases; between extreme examples of which, however, all intervening grades occur.

(i.) In the chronic caseating and ulcerous form the symptoms come on gradually and insidiously. For a period, perhaps, of several months the patient has been gradually failing in health, with loss of appetite and some wasting, together with an irregular pyrexia—a combination of symptoms which, though suggestive of tuberculosis, does not necessarily indicate the abdomen as being specially the seat of the mischief. Soon, however, the patient begins to complain of a feeling of fulness and of vague uneasiness, now and then amounting to actual pain in the belly usually at no special spot, and slight tenderness to touch, sometimes best marked about the umbilicus. The abdomen on examination is found to be somewhat tumid, chiefly from tympanitic distension; but at the same time an irregularly distributed sense of increased resistance is perceived, or it may be that definite evidence of fluid in the peritoneal cavity is forthcoming at a comparatively early stage. As the disease progresses the diffuse pain and tenderness appreciably increase, and an extremely painful sensibility of the skin to the slightest touch is sometimes noticed. The abdomen becomes more and more swollen, in marked contrast to the chest and limbs which emaciate to an extreme degree. The integuments are sometimes dry and harsh, sometimes soft and œdematous, and over the abdomen are not infrequently pigmented — the brownish discoloration being very liable to follow the use of hot fomentations for the relief of the pain. The superficial veins, especially in the lower zone of the abdomen, are apt to be distended and prominent—partly owing to the absorption of the subcutaneous fat which reveals them, and partly to actual hindrance to the return of blood from obstruction in the abdomen. The amount of exudation in this form is seldom very considerable, and what there is shifts with difficulty on altering the position of the patient; it is confined with varying degrees of completeness by adhesions into imperfectly communicating loculi between the coils of intestine or between them and the parietes, giving rise to gurgling sensations on palpation. The physical signs of fluid, therefore, are rarely complete, and its presence is often inferred rather than proved. It is farther noticeable that the amount of fluid varies from time to time very distinctly, now less, now more, without obvious cause. Sooner or later a characteristic doughy sensation with a sense of fixity of the intestines on manipulation may be detected —sensations described with difficulty although recognised easily; and the swelling of the abdomen becomes perceptibly irregular, due in great measure to the formation of the omental and other tumours above described. It is peculiarly characteristic of these lumps, that they are by no means constant in situation, or at any rate that they are not always equally apparent to the observer on inspection or palpation; the varying

degree of distension of the intestines with flatus or fæces, and the irregular and shifting distribution of the fluid in the peritoneal cavity, probably explain this phenomenon. Another sign, and one which when present may almost be regarded as diagnostic of tuberculous peritonitis, is a redness and swelling of the integuments around the umbilicus, followed sooner or later by a discharge of purulent fluid which may become fæcal in character. Various pressure-effects may result from the enlarged lymphatic glands or the omental tumours, such as pain and œdema in the lower extremities; among exceptional manifestations due to the same cause jaundice has been met with, and even a general bronzing of the skin, as recorded by Dr. Colman, brought about by the pressure of swollen glands on the left suprarenal body without implication of its structure.

Passing on to the general symptoms the temperature is very irregular; often remaining for weeks within normal range, it may decline to a daily average of 97° F., or even lower; or it may continue for days or weeks at an elevation of one to three degrees. In the greater number of cases gastric intestinal digestion is very imperfectly performed, as evidenced by the deficient or capricious appetite, the frequent nausea and vomiting, and the marked constipation with occasional outbursts of troublesome diarrhœa, the motions being ill-smelling and of an unhealthy appearance. The tongue is red and irritable or denuded of epithelium. Not infrequently the patient complains of difficulty or even pain in micturition. The pulse is small, quick, and soft, and the whole condition of the patient is one of increasing weakness and exhaustion, preventing any exertion and confining him mostly to bed. The face is pinched and drawn, and there is a progressive anæmia, to which is attributable the œdema about the ankles so commonly noticed. Unless the meninges be involved the intellect remains clear, but the temper is peevish and irritable. Tuberculosis in other organs will necessarily contribute to the sum of the conditions which the patient may manifest; but the abdominal signs when well marked often overshadow the chest disease even though this be well advanced.

Such are the general features and the usual course of an ordinary case of this class. But the exceptions to this description are many. Thus pain, instead of being a prominent symptom, may be almost or quite absent throughout the whole duration of the disease; it may recur periodically with considerable severity, or at other times may scarcely give rise to complaint: the tenderness is probably more usual in its occurrence. Paroxysms of agonising pain, resembling in character biliary or renal colic, have been known to accompany tuberculous enlargement of mesenteric or retro-peritonal glands (1). The extent of emaciation and general malnutrition will obviously be conditioned largely by the degree to which digestion is disturbed and by the coincidence of pulmonary tuberculosis; in some cases the wasting, at any rate in the earlier stages of the disease, may be less considerable than has been described.

The clinical course of those cases of tuberculous peritonitis which are complicated with a tuberculous pleurisy depend somewhat on which

membrane is attacked first. Should it be the pleura there will be the general signs and symptoms indicative of pleuritic inflammation and exudation, usually on one side, though occasionally on both; an interesting case of the latter condition, exceptional in the recovery, is recorded by Dr. Finlay. The chest symptoms may almost completely subside before the evidence of the peritoneal affection appears, or both may coexist; but however this may be, it is the abdominal state which preponderates and mainly contributes to the discomfort of the patient, though an attack of pleurisy may be the immediate cause of death. When the pleurisy is secondary to the peritonitis the former rarely gives rise to prominent symptoms; and the abdominal pain and tenderness with swelling, as well as the diarrhœa and vomiting and other gastro-intestinal symptoms constitute the main disease.

When cirrhosis of the liver complicates tuberculous peritonitis, the disease, as might be expected, runs a more rapid course. A considerable ascites masks the characteristic abdominal features, the effusion frequently being hæmorrhagic. Pain and even tenderness are less marked, the temperature is only sometimes raised above normal, and slight jaundice is present. Pleuritic effusion on one or both sides occurs with tolerable frequency.

As a general rule, cases such as are included within this group run on for a long time, now better, now worse; sometimes even for months the patient improves in health, the symptoms abating and the physical signs becoming less distinctive; then again, without perhaps any apparent cause, the condition becomes more acute in character, a fresh effusion of fluid takes place associated, probably, with a recrudescence of the tubercle, or the gastro-intestinal symptoms increase in severity, and the general nutrition is seriously impaired; meanwhile the caseating and ulcerating processes steadily progress, establishing communications between the intestinal coils, coupled, it may be, with some suppuration, and a hectic temperature or a fæcal fistula; all combining to bring about a fatal result from sheer exhaustion and lack of nourishment. In children especially the remissions of symptoms are conspicuous. Now and then, however, cases are met with, even among those in which the signs and symptoms have been tolerably well marked, when the development of the tubercle appears to be checked, the caseating material to be absorbed, and the inflammatory products to be converted into a fibrous tissue; thus there is a considerable alteration in the clinical aspect of the case, which then comes to correspond to those to be presently described. Owing to this change which frequently takes place in the anatomical characters of the disease, the post-mortem appearances do not always correspond to the indications of the physical signs during life; in this way a very considerable ascites, of the existence of which no doubt could be entertained, might disappear, and leave very little trace after death.

(ii.) The definitely "fibrous" form of tuberculous peritonitis, whether it has been preceded by the acute miliary stage, or has followed the ulcerous variety just mentioned, or whether, as sometimes happens, it

tends towards this form from the first, always runs a very chronic course, and is marked by symptoms which for the most part depend on the mechanical interference with the abdominal functions by the overgrowth of fibrous tissue which to a greater or less degree binds the intestines and viscera into an inseparable mass. The belly is retracted and flattened over the whole or greater part of its area; the costal margin, pubes, and anterior iliac spines prominently project so as to give a hollowed or so-called boat-shaped appearance which is most characteristic; this shape is brought about by the shortened mesenteries and contracted intestines, together with, in extreme cases, some contraction of the solid viscera themselves under the compression of the shrinking fibrous tissue. Pain and tenderness are but slightly marked in such cases, and a persistent and often extreme constipation is the symptom most generally complained of. The impaired function of the gastro-intestinal tract determines a deteriorated nutrition, as shown by the general wasting of the body, which may be considerable. When the fibrosis is more restricted in area, displacement of organs, or adhesions of the stomach and intestine, or compression of the latter by bands may give rise to the most variable symptoms, among which, however, constipation is almost surely prominent, though the general nutrition will suffer less and emaciation may be wanting.

Diagnosis.—From the foregoing account it might be supposed that tuberculous peritonitis is fairly easy of recognition, but this is oftentimes far from the case. Well-marked cases are doubtless diagnosed without much difficulty provided a reliable history be obtainable; but a very large proportion of the cases, whether in child or adult, are not well marked, and the diagnosis may long remain in doubt; whilst now and then in the absence of signs and symptoms during life the existence of the disease is first disclosed at the post-mortem examination.

Excluding those cases which manifest no abdominal symptoms, and where the features of a generalised tuberculosis are not specially manifested in any one part, and putting aside also those cases where the symptoms referable to the chest or cranium so predominate over those of the abdomen that these latter can be scarcely said to exist, it remains to distinguish acute abdominal tuberculosis from chronic tuberculous peritonitis of the ulcerous and fibrous varieties.

The similarity of acute tuberculosis of the peritoneum to enteric fever has been already referred to. The malaise and sense of illness, the elevation of temperature and the diarrhœa, often with pea-soup stools, are features possessed by the two maladies in common; and the resemblance is heightened still further by the occasional existence of a little bronchitis and even of headache in the serous affection. The absence of rash, so far as it goes, is in favour of typhoid fever; whilst the diazo reaction of the urine is too uncertain to be of much value. Probably the most trustworthy distinctions are to be found in the limitation of pain and tenderness to the cæcal region in enteric fever, and in the temperature curve. The progressive rise for the first few days, followed by a persistent uniformity for about a fortnight, a course more or less distinctly characteristic

of typhoid fever, is rarely if ever to be seen in acute tuberculosis ; in this disease the temperature may be at its maximum within the first few days, or the highest point may not be reached for several weeks ; whilst, if the chart for any long period be available, a far greater irregularity will generally be observable in the peritoneal disease. With a careful record for a month there should rarely be any doubt, but for a fortnight, or even more, the conclusion may be in suspense. Reference has been made in tuberculosis to the occasional recurrence of maximum daily temperature in the earlier part of the day, and this is seldom the case in typhoid fever. Widal's test (vol. ii. p. 1145) will probably prove very useful in distinguishing these diseases.

For the recognition of chronic tuberculous peritonitis it is desirable to keep in mind the descriptions of typical forms such as have been set forth. But it is most needful to emphasise the fact that many cases do not conform thereto. The irregularity of the course of the disease, the frequent intermissions with marked improvement, the exacerbation of symptoms for lengthened periods, the degree of illness of the patient, often out of proportion to the physical signs which may be obscure and far to seek, and the prolonged duration of the malady—indefinite as these characters may be—are often those alone upon which the diagnosis can be based ; and for their appreciation time is requisite.

Certain specific features of the disease may add to the difficulties of diagnosis. Thus encysted collections of fluid, with induration and matting together of the structures confining it, may simulate ovarian cysts, and nothing short of a laparotomy may settle the question ; the same may be said for the omental tumours which have been frequently mistaken for malignant growths of the peritoneum, or even of the colon or ovary, the disturbances of the bowels or extreme constipation favouring the error. As a rule the position of these masses below the line of resonance furnished by the transverse colon should distinguish them from hepatic enlargements which lie above that line, and their irregular shape should prevent their being mistaken for the spleen, the outline of which, with its characteristic notch, is usually well preserved in the various morbid changes which that organ may undergo. Dr. Osler attributes some importance to the frequently prolonged subnormal temperature of many cases of omental tuberculous tumours as a character serving to distinguish them from growths of a malignant nature.

The diagnostic value of redness and induration around the umbilicus and the discharge of pus or fæcal matter therefrom has been mentioned.

The examination of the fluid withdrawn from the abdomen is not very helpful. The presence of the B. tuberculosis would be conclusive, but they are rarely to be found, and their absence does not exclude the existence of tubercle. Inoculation experiments, however, may sometimes determine the nature of the disease.

The distinction during life between a tuberculous and a simple peritonitis must often remain uncertain, if the existence of the latter condition be admitted. Now that it is known that a very definite tuber-

tends towards this form from the first, always runs a very chronic course, and is marked by symptoms which for the most part depend on the mechanical interference with the abdominal functions by the overgrowth of fibrous tissue which to a greater or less degree binds the intestines and viscera into an inseparable mass. The belly is retracted and flattened over the whole or greater part of its area; the costal margin, pubes, and anterior iliac spines prominently project so as to give a hollowed or so-called boat-shaped appearance which is most characteristic; this shape is brought about by the shortened mesenteries and contracted intestines, together with, in extreme cases, some contraction of the solid viscera themselves under the compression of the shrinking fibrous tissue. Pain and tenderness are but slightly marked in such cases, and a persistent and often extreme constipation is the symptom most generally complained of. The impaired function of the gastro-intestinal tract determines a deteriorated nutrition, as shown by the general wasting of the body, which may be considerable. When the fibrosis is more restricted in area, displacement of organs, or adhesions of the stomach and intestine, or compression of the latter by bands may give rise to the most variable symptoms, among which, however, constipation is almost surely prominent, though the general nutrition will suffer less and emaciation may be wanting.

Diagnosis.—From the foregoing account it might be supposed that tuberculous peritonitis is fairly easy of recognition, but this is oftentimes far from the case. Well-marked cases are doubtless diagnosed without much difficulty provided a reliable history be obtainable; but a very large proportion of the cases, whether in child or adult, are not well marked, and the diagnosis may long remain in doubt; whilst now and then in the absence of signs and symptoms during life the existence of the disease is first disclosed at the post-mortem examination.

Excluding those cases which manifest no abdominal symptoms, and where the features of a generalised tuberculosis are not specially manifested in any one part, and putting aside also those cases where the symptoms referable to the chest or cranium so predominate over those of the abdomen that these latter can be scarcely said to exist, it remains to distinguish acute abdominal tuberculosis from chronic tuberculous peritonitis of the ulcerous and fibrous varieties.

The similarity of acute tuberculosis of the peritoneum to enteric fever has been already referred to. The malaise and sense of illness, the elevation of temperature and the diarrhœa, often with pea-soup stools, are features possessed by the two maladies in common; and the resemblance is heightened still further by the occasional existence of a little bronchitis and even of headache in the serous affection. The absence of rash, so far as it goes, is in favour of typhoid fever; whilst the diazo reaction of the urine is too uncertain to be of much value. Probably the most trustworthy distinctions are to be found in the limitation of pain and tenderness to the cæcal region in enteric fever, and in the temperature curve. The progressive rise for the first few days, followed by a persistent uniformity for about a fortnight, a course more or less distinctly characteristic

of typhoid fever, is rarely if ever to be seen in acute tuberculosis; in this disease the temperature may be at its maximum within the first few days, or the highest point may not be reached for several weeks; whilst, if the chart for any long period be available, a far greater irregularity will generally be observable in the peritoneal disease. With a careful record for a month there should rarely be any doubt, but for a fortnight, or even more, the conclusion may be in suspense. Reference has been made in tuberculosis to the occasional recurrence of maximum daily temperature in the earlier part of the day, and this is seldom the case in typhoid fever. Widal's test (vol. ii. p. 1145) will probably prove very useful in distinguishing these diseases.

For the recognition of chronic tuberculous peritonitis it is desirable to keep in mind the descriptions of typical forms such as have been set forth. But it is most needful to emphasise the fact that many cases do not conform thereto. The irregularity of the course of the disease, the frequent intermissions with marked improvement, the exacerbation of symptoms for lengthened periods, the degree of illness of the patient, often out of proportion to the physical signs which may be obscure and far to seek, and the prolonged duration of the malady—indefinite as these characters may be—are often those alone upon which the diagnosis can be based; and for their appreciation time is requisite.

Certain specific features of the disease may add to the difficulties of diagnosis. Thus encysted collections of fluid, with induration and matting together of the structures confining it, may simulate ovarian cysts, and nothing short of a laparotomy may settle the question; the same may be said for the omental tumours which have been frequently mistaken for malignant growths of the peritoneum, or even of the colon or ovary, the disturbances of the bowels or extreme constipation favouring the error. As a rule the position of these masses below the line of resonance furnished by the transverse colon should distinguish them from hepatic enlargements which lie above that line, and their irregular shape should prevent their being mistaken for the spleen, the outline of which, with its characteristic notch, is usually well preserved in the various morbid changes which that organ may undergo. Dr. Osler attributes some importance to the frequently prolonged subnormal temperature of many cases of omental tuberculous tumours as a character serving to distinguish them from growths of a malignant nature.

The diagnostic value of redness and induration around the umbilicus and the discharge of pus or fæcal matter therefrom has been mentioned.

The examination of the fluid withdrawn from the abdomen is not very helpful. The presence of the B. tuberculosis would be conclusive, but they are rarely to be found, and their absence does not exclude the existence of tubercle. Inoculation experiments, however, may sometimes determine the nature of the disease.

The distinction during life between a tuberculous and a simple peritonitis must often remain uncertain, if the existence of the latter condition be admitted. Now that it is known that a very definite tuber-

culous affection may subside to a great extent, and leave no indication of tubercle, the diagnosis of "simple" cases will be less frequently made. But so far as physical signs are concerned no distinctive difference is to be expected, since the effusion and inflammatory new growth must give rise to the same objective conditions in both groups of cases, whilst the history and general clinical course too often furnish but indefinite data for a judgment. Valuable information may be derived from the existence of tubercle elsewhere but, in the frequent cases in which the new growth is restricted to the abdomen, the difficulty of distinction may be insuperable.

Prognosis.—Tuberculous peritonitis is certainly not the invariably hopeless disease it was once thought to be; and, although it still determines a considerable mortality, the very distinct benefit which follows treatment in a large number of cases has largely diminished its hitherto fatal character. Moreover, the recognition of the fact that tubercle, in a certain proportion of even well-marked cases, tends to its own cure by undergoing fibroid transformation has rendered the prognosis more hopeful; though it is true that the prolonged life may not be a completely healthy one, owing to the permanent ill effects brought about by the development of fibrous tissue within the abdominal cavity, and the oftentimes grave interference with the free movement of the contained viscera. As compared to tubercle in other regions, a larger proportion of cases of the peritoneal affection tend to improvement, whether spontaneously or by treatment, than can as yet be affirmed of cerebral or even of thoracic tuberculosis. In forecasting the course of a given case, the liability to improvement, even for months, with a subsequent recurrence of symptoms and fatal result, must not be forgotten. On the other hand, the existence of pulmonary tuberculosis, or pleurisy, or cirrhosis of the liver, or tuberculous ulceration of the intestine, would very seriously affect the prognosis. The longer the case has lasted the longer it may last, and improvement may take place even in the most unlikely; a persistent diarrhœa is the symptom most to be feared. In proportion to the extent of the fibroid change in the tubercle and the absence of caseous degeneration, and particularly of an ulcerous process, the prognosis may be regarded as favourable; but the grounds for anticipating the one or the other of these conditions are not yet formulated.

Treatment.—In tuberculous peritonitis, no less than in tuberculosis elsewhere, much may be hoped for from such hygienic measures as are comprised in the avoidance of infection by food—milk, meat and meat juice—or air, a subject already treated in the article on "Tuberculosis."

What has been said in the foregoing section on the medicinal treatment of chronic peritonitis is equally applicable to the tuberculous, and perhaps with more confidence. Applications to the surface of the abdomen for the relief of pain, and for promoting the absorption of the inflammatory products, and drugs for the attendant constipation and the maintenance of the general health, are as suitable in the one case as the other, the indications for treatment being substantially identical. Special benefit in children has been claimed for the administration of creasote,

whether by the mouth in milk, or combined with cod-liver oil as a daily enema (21).

Since 1862, when Sir Spencer Wells opened an abdomen for the removal of a supposed ovarian tumour and found a large quantity of opalescent fluid and a peritoneum studded with tubercles, a simple laparotomy has been frequently performed for the relief of tuberculous peritonitis and often with the same satisfactory result, which in that case was cure; and in the great majority of cases very considerable benefit has ensued. A large mass of evidence from all quarters is now available, which is unanimous in establishing the procedure as not only justifiable, but in many cases imperatively called for.

The variation in the anatomical characters no less than in the clinical features of the disease has given rise to some difference of opinion, as might reasonably be expected, as to the most suitable cases for operation as well as concerning certain details of the operation itself; but all are agreed in the benefit which follows its performance.

That the expression "cure," as opposed to mere "relief," is in a certain proportion of cases justifiable, follows not only from the fact that numberless instances are recorded in which well-marked and even severe symptoms of tuberculous peritonitis existed and abundant tubercles were disclosed at the operation, and yet recovery from all discomfort and absence of further symptoms for many years resulted, but still more conclusively also from the well-authenticated cases recorded by many surgeons (2, 10, 13, 19), who have found that on a second laparotomy, performed some months later for some other reason, the tubercles, which were but too obvious at the first operation and were proved to be such both microscopically and by inoculation, had entirely disappeared.

Concerning the operation itself, it would certainly seem that a mere tapping of the abdomen by an aspirating needle or small trocar is insufficient; there must be a considerable opening and a free evacuation of any fluid present. Beyond this nothing can be said to be necessary. Washing out the peritoneal cavity with sterilised water, warm boracic lotion, hot solution of creolin or other disinfectants, dusting with iodoform, or the withdrawal of only a small quantity of fluid and injection of camphorated naphthol into the cavity, have all been practised and warmly advocated; but, as experience has shown, with no greater advantage than is obtained by a simple opening, free drainage and closure of the wound without flushing or other manipulation.

A slight rise of temperature has been commonly noticed after the laparotomy, lasting some hours or even a day or two; but as a rule recovery from the operation (which has of course been carried out with all antiseptic precautions) is rapid, and improvement in the condition of the patient is soon apparent. Some reaccumulation of fluid requiring withdrawal is occasionally met with.

It may sometimes be necessary to remove certain structures, such as the Fallopian tubes or mesenteric glands, which are extensively infiltrated; this apparently can be done without increasing the risk of the operation.

Every form in which tuberculous peritonitis occurs has been made the subject of operative treatment, and with benefit. Undoubtedly the most promising cases are those in which there is effusion, whether serous, seropurulent, or purulent—free or encysted; and in such cases laparotomy may almost be regarded as the only proper treatment. Even in the acute cases of peritoneal tubercle accompanied by much ascites the procedure may be resorted to, although the result is not altogether so satisfactory as in the more chronic forms. Nor does the presence of tubercle in the lungs, unless far advanced, offer a contraindication (20), temporary benefit at least may be expected. Notwithstanding the risks attendant on opening the abdomen when there is little or no effusion, and merely a condition of widespread fibrous adhesion among the viscera—risks due to wounding the bowel or inflicting damage by tearing away the adhering bands—yet very marked improvement has been recorded as having followed a simple incision through the abdominal wall and closing up the wound. Operation in such cases, however, is scarcely to be undertaken except with a view to relieve intestinal obstruction.

The statistics compiled by different authors fully justify the recourse to operation which with proper precautions is scarcely attended with danger. Thus König collected the records of 131 cases in which laparotomy had been performed for tuberculous peritonitis, including 14 operated on by himself; of these 120 were women and 11 men; 70 per cent being over 20 years of age. In 107 of these the operation was satisfactory, being completely so in 84, or 65 per cent; whilst deaths directly following the operation only amounted to 3 per cent. Lindner collected 205 cases, of which 7·5 were fatal from the operation, the causes being collapse, sepsis, or acute peritonitis. In only 186 of these was the sex recorded, and of them 11·3 per cent were males. Rorsch in 1893 found in 358 cases, the records of which he had collected, a mortality of 5·59 from the operation, and complete recovery in 250, being about 70 per cent. Less successful results were obtained by Frees, who only claimed complete success in 5 of 18 cases treated by himself, whilst in 12, though no ill effect was attributable to the operation, no benefit followed. All these patients were females—in some there were complicating conditions connected with the uterus or appendages.

It may be observed that the great preponderance of females in the statistics dealing with operation in this affection is to be explained by the great frequency with which the Fallopian tubes are the seat of tubercle, and by the performance of laparotomy in a very large number of cases to relieve a supposed tumour or cyst connected with the reproductive organs; the nature of the disease only being disclosed by the operation. It is not to be supposed that females are more liable to peritoneal tuberculosis, for if there be a difference the reverse would be a truer statement.

Various are the suppositions to explain the effect of this treatment, but it cannot be said that any are wholly satisfactory. The facts clearly suggest that peritoneal tubercle is not possessed of great activity or powers of resistance, and that its natural tendency to obsolescence is

readily facilitated by what appear to be but very trifling disturbances of the conditions under which it is developing. Bumm and others have demonstrated that after a simple laparotomy the gray granulations undergo round cell infiltration and cicatricial changes, with disappearance of the epithelioid and giant cell elements of the tubercle. Kischenski also showed this to be the case by experiments on guinea-pigs. The exact determining factor of this change is the point at issue. The generally credited cause is the removal of the fluid, but it is doubtful if this be the sole cause. With less reason, the admission in the course of the operation of air, or of light (14), or of putrefaction bacteria whereby toxalbumins destructive of the tubercle bacillus are formed (16), have been regarded as the causes; and Dr. Duran has recommended, in place of laparotomy, simple paracentesis abdominis, withdrawal of fluid and injection into the peritoneal cavity of dry aseptic air. The bacillus tuberculosis, being aerobic, the asserted bactericidal effect of free admission of air is not easy to explain; nor is it likely that in ordinary operations either air or light penetrates very far into the abdominal cavity, although direct sunlight is known to attenuate the virulence of the organism of tubercle, or even to destroy it. Irrigation with antiseptic solutions seems a more potent means, but it is by no means essential to the attainment of the desired result.

W. H. ALLCHIN.

REFERENCES

1. BIER, A. *Deut. med. Woch.* No. 23, 1892.—2. BRUHL. "The Treatment of Tuberculous Peritonitis," *Gazette des hôpitaux*, Oct. 1890.—3. BUMM. "On the Cure of Peritoneal Tuberculosis after Laparotomy," *Centralbl. f. Gynäk.* No. 22, 1893.—4. CARR, J. WALTER. "The Starting-Points of Tuberculous Disease in Children," *Trans. Med. Soc. Lond.* 1894, vol. xvii. p. 288.—5. COLMAN, W. S. "The Distribution of Tubercle in Abdominal Tuberculosis," *Brit. Med. Jour.* 1893, ii. p. 740.—6. "Discussion on Abdominal Tuberculosis in Childhood," *Brit. Med. Jour.* 1893, vol. ii. pp. 734-740.—7. DURAN. "Treatment of Tubercle of the Peritoneum," *L'Union Médicale*, Sept. 30, 1893.—8. FINLAY, D. "Case of Tubercular Peritonitis and Double Pleurisy in Youth—Recovery," *Clin. Soc. Trans.* Oct. 24, 1890.—9. FREES, G. "The Operative Treatment of Tuberculous Ascites," *Deut. med. Woch.* Nov. 8, 1894.—10. KEETLEY, C. B. "Three Cases of Tubercular Peritonitis in which Abdominal Section was performed," *Brit. Med. Jour.* 1890, vol. ii. p. 1028.—11. KISCHENSKI. *Centralbl. f. allgem. Path.* Nov. 15, 1893.—12. KÖNIG. "Peritoneal Tuberculosis and its Cure by Laparotomy," *Centr. f. Chir.* 1890, p. 657.—13. KÜMMILL. "Laparotomy in Peritoneal Tuberculosis," *Wien. med. Woch.* Feb.-March, 1888.—14. LAUENSTEIN. "Observations on a remarkable Cure of Peritoneal Tuberculosis by Laparotomy," *Centralbl. f. Chir.* No. 42, 1890.—15. LINDNER. "On the Operative Treatment of Peritoneal Tuberculosis," *Deut. Zeitschrift f. Chir.* 1892, Bd. xxiv. p. 448.—16. MORRIS, R. T. "The Reasons why Patients Recover from Tuberculosis of the Peritoneum after Operation," *Phil. Med. News*, October 13, 1894.—17. OSLER, W. "Tubercular Peritonitis," *Johns Hopkins Hospital Reports*, vol. ii. No. 2, 1890.—18. POPPERT. *Münch. med. Woch.* Aug. 23, 1892.—19. RICHELOT. "On Tuberculous Peritonitis," *Bull. et mem. de la Soc. de Chir.* 1892, p. 312.—20. SYMS, PARKER. "The Influence of Laparotomy upon Tuberculosis of the Peritoneum," *New York Med. Jour.* Feb. 7, 1891.—21. THOMER, Prof. "Creosote in the Treatment of Pleuro-peritoneal Tuberculosis in Children," *Lancet*, June 17, 1897.—22. WOODHEAD, SIMS. "Tabes Mesenterica and Pulmonary Tuberculosis," *Reports of Laboratory of Royal College of Physicians, Edinburgh*, vol. i. 1888.—23. WAGNER, E. "The Concurrence of Cirrhosis of the Liver and Tuberculous Peritonitis," *Deutsch. Archiv f. klin. Med.* Bd. xxxiv. 1884.

W. H. A.

New Growths of the Peritoneum

The serous membrane which lines the abdominal cavity, and forms a more or less complete covering to the viscera contained therein, is continuous by its outer surface with the subperitoneal connective tissue; in amount this tissue varies very considerably in different situations, being scarcely present over the surface of such organs as the liver and spleen, and very abundant over the posterior regions of the abdomen. Owing to this continuity of structure it is undesirable, either clinically or from an anatomical standpoint, to attempt to distinguish the new growths which are essentially peritoneal from those which are sub- or retroperitoneal in position. But neoplasms which originate in the organs placed outside the peritoneum—such as the lymphatic glands, the kidneys, the suprarenal capsules, or in parietal structures such as the spinal column and trunk muscles—are obviously not included within the scope of peritoneal growths.

The histological elements of the peritoneum and its subjacent tissue are as follows: first, a layer of endothelium; this is superimposed on a more or less distinct basement membrane consisting of a ground substance supported by a reticular framework of white and elastic fibres, which merges into the underlying areolar tissue; within the meshes of this framework, both retroperitoneal and between the layers of the mesenteric and omental folds, is a very variable quantity of fat cells; and together with these are some blood-vessels, plain muscular tissue, nerves, and lymphatics, these last communicating by stomata with the cavity of the serous sac. It is evident that these elements mainly belong to the connective tissue series, and that in consequence the majority of new growths will be of this origin; how far any can be regarded as epithelial in character will be presently discussed. In accordance with this fundamental affinity it comes about that the tumours met with seldom consist wholly of one tissue, whether fibrous, adipose, plain muscular or sarcomatous (embryological); they commonly present indications of more than one of these structural elements, and that which preponderates gives the name to the growth.

As compared with the other serous sacs—pleuræ and pericardium—tumours of the peritoneum and subperitoneal tissue are more frequent; but even here they are far from common, and in respect to those of a malignant character there is much yet to be learned.

Fibroma.—Disseminated fibroid nodules, varying in size from a millet seed to a split pea, are occasionally met with over the peritoneum—parietal, mesenteric and visceral. They may be many in number, or but two or three. Not unfrequently they represent tuberculous granulations which have undergone fibrous involution; but some may be primary new growths of fibrous tissue. "The fibromata merge," says Mr. Anderson, "on the one hand into the lipomata, on the other into the

myomata; and it is probable that many of the older reports of fibrous tumours really refer to myomata. Purely fibrous growths may, however, develop in the subperitoneal tissue, or may have arisen in the first instance from the symphysis pubis, or other parts of the fibrous tissues of the abdominal pelvic wall, and have become detached into the subperitoneal interspaces" (3). Pure fibromata have seldom been known to attain any considerable size, and when small give rise to no symptoms leading to their detection during life.

Lipoma. Fibro-lipoma.—Localised overgrowths of adipose tissue—frequently with some relative excess of fibrous tissue—are among the most frequent of peritoneal and retroperitoneal neoplasms. The conditions determining their origin are quite unknown, but they seem to be distinct from those which lead to that diffuse development of fat which we know as general obesity. These growths are of far more frequent occurrence in women than in men, in the proportion of 25 to 16 (1); with rare exceptions they have been met with in adults only.

The subserous adipose tissue in the neighbourhood of the kidneys and iliac fossa—with a slightly greater frequency on the right side—is the usual situation from which these tumours spring, and the majority of them continue to be retroperitoneal; from this position, however, while retaining more or less completely a connection with the seat of origin, they may penetrate between the layers of the omentum or mesentery. More rarely definite tumours of this nature originate in the subperitoneal tissue of the mesenteric or omental folds, where a general fatty overgrowth in varying degree is frequently observed. Mr. Treves (24) records a case of a lipoma weighing 4½ lbs. which he removed from between the layers of the broad ligament, a very exceptional position, and one where adipose tissue is rarely if ever seen, even in extreme obesity. Two similar cases have been met with on post-mortem examination. To be included within this category are the fatty tumours which correspond with extremely enlarged appendices epiploicæ. Springing from extremely slender or broadly sessile attachments, they have been met with completely detached and lying free in the serous sac, although it is very doubtful whether the separation occurred long before death.

An exceedingly interesting and, from a surgical point of view, most important group of subperitoneal lipomata are those which extend outside the abdominal cavity at the inguinal or femoral rings or in the linea alba, where they closely simulate omental herniæ. They have been known to attain enormous size and a weight of many pounds; and containing, as they frequently do, more or less extensive prolongations of the peritoneum, they may, even when very small, be the cause of pain, vomiting, and other symptoms which are relieved by their removal.

The greater number of cases have been met with between the ages of thirty and fifty; but children or indeed infants are not wholly free. Lebert records the removal of a fatty tumour which occupied the left side of the abdomen and had grown with the growth of the patient since it

was first noticed at the age of six months; and Lauwers records one, removed at the age of seven years, from a child in whom it had been recognised since a fortnight after birth; its weight was 6 lbs., almost a third of the total weight of the child after its removal. Cima has recently described another in an infant aged twenty-two months.

It is rare to find more than one of these growths, although the co-existence of several is not entirely unknown (10). As the masses are often multilobed, the portions being partially separated, it is possible that some of the largest of these tumours have been formed by a fusion of several at first distinct.

There is great variation in their size which, so far as the clinical histories show, is not always proportionate to their recognised duration. Several have been recorded weighing upwards of 55 lbs. Masses of this bulk have appeared to fill the abdominal cavity completely, displacing the intestines to the sides; if, however, the mass have originated behind the peritoneum, either in the perirenal tissues or at the root of the mesentery, some part of the colon, usually the ascending or descending, may be stretched over the front of the tumour. This is not the case when the growth is developed in the omentum or mesentery.

In structure these growths exhibit much difference in the relative proportions of the connective tissue and fat cells of which they are composed, the former material sometimes being very considerably in excess. As a rule, being less vascular, they are firmer and paler than healthy subcutaneous adipose tissue; the perirenal fat, it may be observed, normally tends somewhat in the same direction.

The majority of tumours of this class, however, exhibit a greater or less proportion of other material; thus besides the fibroid tissue, mucoid or myxomatous tissue is of frequent occurrence, so that a myxo-lipoma is almost as common as a pure lipoma. Cartilage has very rarely been found in association with a fibro-lipoma. Areas of calcareous degeneration—that frequent sequence of fatty change—are sometimes seen, and even true bone—osteo-lipoma. It is possible, however, that these last are to be regarded as fœtal inclusions in which the other tissues have undergone fatty change, rather than true lipomata in which bone has developed itself. Sometimes certain parts of the mass exhibit all the microscopic appearances of sarcoma which is both histologically and genetically a term of the connective tissue series. Whilst some exhibit a very considerable degree of capsulation, and are "shelled out" with comparative facility, others blend in their attachments with the subserous adipose tissue from which they spring, and are marked off by no definite limits.

The *physical signs* of these tumours and the *symptoms* to which they give rise are seldom very distinctive, and their nature is rarely diagnosed. Usually they have been mistaken for ascites, or for ovarian cystic tumours; and paracentesis abdominis has been frequently performed for their relief without result. Nor is the error to be wondered at. When of large size and distending the abdomen from pelvis to thorax, chiefly

in an antero-posterior direction rather than laterally, their soft doughy feel with an indistinct sense of fluctuation may easily be confounded with fluid, either encysted or free. If there be resonance in one or both flanks the signs of ovarian cyst are closely simulated, whilst, should the flanks be dull, the condition might be explained by a large collection of free peritoneal fluid. When there is an area of resonance in front of the mass, due to an adherent colon, and dulness reaches into one flank, the growth may with reason be considered as connected with the kidney, from the adipose tissue around which organ it may have developed. The smaller growths are freely movable, for the most part painless, and give rise to no symptoms; and even with the largest masses little beyond a sense of fulness and weight is complained of. Some compression of the mesenteric and other vessels may be induced by the growth, and even ascites subsequent thereto has been noticed; but there is very seldom any peritonitis. As a rule the patients are in fairly good general health and well nourished; but in cases attended with much emaciation a diagnosis of malignant disease has been made, and any attempt at relief by operation refused on this ground. When the tumours have undergone myxomatous change, and still more when the fatty growth is complicated with sarcoma, the masses tend to grow with considerable rapidity and to interfere the more with the general nutrition of the patient; so that a diagnosis of malignancy is increasingly probable (18). The surface of the mass is usually smooth, though sometimes it is deeply lobulated; it is but little tender on handling, if at all. When of considerable size symptoms due to pressure on the intestinal canal or on the bladder, or to interference with the free action of the diaphragm, may arise and attain to a severe degree; more often œdema of the lower limbs occurs, sometimes most marked on the side from which the tumour sprang.

The data on which a *diagnosis* may be made is to be gathered from the foregoing description of the signs and symptoms. The difficulty is much greater in women, owing to close resemblance of these masses to growths in connection with the pelvic generative organs.

Although in the main of a simple kind the *prognosis* of these growths is far from being very favourable unless recognised and removed while of small size. The comparatively slight risk of a laparotomy nowadays will doubtless permit us to obtain better results than hitherto, since in most of the recorded cases the tumours have been regarded as too large and too formidable for operation; or, when this has been attempted, their large area of attachment has frequently prevented any successful interference. Of the 42 cases collected by Prof. Adami (1), in 26 "the tumour was removed wholly or almost wholly. In 12, or 46·1 per cent, the operation was successful." The same writer draws especial attention to the danger due to the position of the colon in front of the tumour, and "as a consequence unless great care be taken in the removal, the blood-supply of this portion of the intestine is cut off and gangrene or necrotic inflammation ensues, which seems to have been the history in most of the fatal cases."

Fibro-myoma. Myoma.—Rare cases of new growth originating in the retroperitoneal tissue and composed of fibrous tissue with a variable amount of plain muscular fibre cells, have been placed on record. They correspond in structure to the frequently occurring fibro-myomata of the uterus, and with this organ some of those which extend into the peritoneal cavity are primarily connected, retaining their covering of serous membrane. In a few situations, such as the uterine ligaments and the root of the mesenteries as well as the sustentacular ligaments of the peritoneum, a small amount of plain muscular tissue is normally to be found in the subserous connective tissue, and it is from this source that these tumours arise. A large example of this variety of growth weighing upwards of nine pounds (after the removal of a large quantity of œdematous fluid) has lately been recorded by Mr. Shield, and a most interesting case of multiple fibro-myomata, developed in a girl of nine years of age who was admitted to the Tokyo Hospital, Japan, is described by Mr. Anderson. In this case "twenty-one rounded tumours of various sizes, the heaviest of which weighed over 7 lbs., were removed from different parts of the subperitoneal tissue, extending from the pelvis to the hypochondrium. They were distinctly encapsulated and shelled out readily." Like the mass removed by Mr. Shield they are described as having been "soft but elastic, white and wax-like on section." Of eleven cases of fibro-myomata of the broad ligament—one of the more usual situations for their development—collected by Mr. Bland Sutton, the majority weighed upwards of 16 lbs., and all occurred in women over thirty-five years of age. So far as is known these growths exhibit no peculiarities, serving to distinguish them from the fibro-lipomata, until they are submitted to microscopical examination; and the signs and symptoms to which they give rise correspond to what has been already stated. Those situated in the broad ligament may cause considerable displacement of the uterus and ovaries, and, after remaining stationary for a long period, are apt to take on a rapid rate of growth.

Sarcoma.—Among the new growths of the peritoneum and subperitoneal tissue which exhibit the clinical features of malignancy those presenting the microscopical characters of sarcoma are the most frequent. Not that it is to be inferred, however, that they are of common occurrence, but only relatively so in comparison with other peritoneal tumours. It is probable that most of the cases formerly recorded as "peritoneal cancer" were really examples of sarcoma.

Their most usual situation is in connection with the retroperitoneal connective tissue; less commonly they involve the great omentum; still less often they develop in one of the mesenteric folds, and, very exceptionally, in the broad ligament. In many cases the growth appears to have arisen primarily in one or other of these sites; but even then smaller masses are often to be met with apart from the main tumour, especially with those more distinctly peritoneal — the nodules being scattered about on other parts of the serous membrane and sometimes on

the surface of the liver, where they are often umbilicated. Occasionally the retroperitoneal lymphatic glands are affected, but very rarely any other organs, whether abdominal or thoracic. In those patients in whom there is reason to believe the growth to be secondary, the testis or ovary appears to be the usual primary seat (16, 21). However extensive they may have become, even to the enveloping of such organs as the kidney and pancreas, they exhibit but little tendency to invade these viscera, although extension into the lumen of the intestine has been met with (17).

Microscopically these growths present the ordinary appearance of round-celled or spindle-celled tumours, or the variety denominated lympho-sarcoma; they vary much in degree of hardness in accordance with the proportion of fibroid elements and the extent of degenerative changes they may have undergone, for it is common to find in the larger masses, which may attain a bulk of thirty pounds weight and upwards, an extensive conversion into myxomatous tissue, or even into cysts; and reference has been already made to the association of sarcoma with fatty overgrowths. Occasionally the mass is extensively infiltrated with pigment which is sometimes derived from blood that has been effused into the tumour; for these neoplasms are generally very vascular, the softer, so-called "encephaloid" specimens being especially so. An extremely interesting feature presented by some of them is that of a "mixed" growth, in which the histological features of sarcoma and carcinoma are associated (2). Unlike the lipomata, these tumours are of more frequent occurrence in males, and although usually met with in adults are not unknown at an early age. A doubtful history of injury is sometimes obtainable.

It is not unusual to find the peritoneum in the immediate neighbourhood of these growths exhibiting a moderate amount of chronic inflammatory change whereby adhesions of varying firmness are established between the mass and adjacent structures, especially to the parietal layer of the serous membrane; but the peritonitis is strictly limited to the parts concerned, and is quite secondary and subordinate. Contrasted with the non-malignant growths in this region the sarcomata interfere with the abdominal circulation far more seriously, as may be seen by the great distension of the superficial veins, and also by the more frequent presence of an ascites, which may be considerable, or of an œdema of one or both lower limbs.

Signs and symptoms.—Apart from the manifestations of general malnutrition, wasting, anæmia and failing strength, which are determined by these growths wherever situated, there will be special signs and symptoms more or less distinctive in character, due to their size and position, and to this extent available for diagnostic purposes.

The retroperitoneal tumours, in their extension forwards, of necessity tend to displace the kidneys, pancreas and intestines laterally, as do the previously described solid growths in this situation; like them also they frequently retain the descending colon in close adherence to their anterior

surface, a circumstance that explains the variable character of the percussion note obtainable over the most prominent part of the mass, according as the bowel is or is not distended with gas. Owing to the more extensive attachment, and the situation from which they spring, these growths are more fixed than the other varieties and move but little under manipulation.

The omental sarcomata for the most part form hard, flattened, smooth or slightly irregular masses, sometimes appearing to consist of two or more separated portions, scarcely if at all tender and seldom painful, situated mainly to one side of the middle line, and rather oftener to the right below the level of the liver from which they are more or less distinctly separated by a band of resonance due to the transverse colon. The lower margin is plainly to be felt as a thin edge, and the whole tumour, which is freely movable, especially from side to side, feels quite superficial and is uniformly dull; unless on deep percussion when a subresonant note is elicited. Such a tumour, says Mr. Treves (25), "is one of the most characteristic of abdominal swellings, and feels like a cake of no great thickness moulded to the curve of the abdominal wall."

Those sarcomata which grow in the mesentery, like the preceding, are very movable unless the parietal adhesions be extensive; and they preferably occupy a position in the middle line just above or below the umbilicus. In shape they tend to be globular rather than flat and, felt through the coils of intestine before they are sufficiently large to reach to the surface, they have much of the character of a tense, thick-walled cyst.

Coincident with his discovery of the abdominal tumour, or preceding it, the patient may complain of vague abdominal uneasiness with a general sense of ill health, colicky pains, nausea, sickness of an irregular character and loss of appetite, and of that sense of weight and sinking so commonly associated with abdominal disturbance with or without a tumour. Such symptoms which are in no sense characteristic, and are apt to be set down to a gastro-intestinal indigestion, tend to progress in spite of remedies until the recognition of the tumour explains the condition. Gradually a deep-seated pain in the belly, perceptible more "from the front" than "from the back," as the patient may express it, becomes a marked feature in the retroperitoneal growths; or it may be that for this symptom relief is sought before the detection of a tumour. Later, as the growth becomes larger and more perceptible, various pressure results, especially referable to the bowels, are liable to occur; and constipation amounting to almost complete obstruction may be established.

The *diagnosis* of these growths from other intra-abdominal abnormalities is a matter of considerable difficulty. The largest and softest varieties, like the lipomata, closely simulate an ascites, especially if there be an area of resonance in front of the mass and the flanks are dull; whilst the coexistence of fluid in the peritoneum, giving rise to unmistakable signs, may favour the supposition.

When a definite solid tumour can be detected various are the conditions for which it may be mistaken. Of these a fæcal accumulation may closely resemble an omental growth; and it may not be until after several large enemas have been administered (which in all such cases should, unless good reason to the contrary, be an invariable preliminary) that what is fæcal can be distinguished from what is not. An educated touch may detect a greater hardness and uniformity in the growth than in the scybalous masses, but this quality, even if present, is not always to be relied upon, and tenderness may accompany or be absent from each disorder.

Probably in women sarcoma is most often confounded with cystic or semi-cystic growths of the ovary or broad ligament; and a vaginal examination should not be omitted, though, of course, the result may be negative.

Both the retroperitoneal and omental growths are apt to be mistaken for renal tumours, and the relation of the dull to the resonant areas favours the error. When the mass is movable and of no great size its greater range of displacement from side to side soon helps to distinguish it from a movable kidney, the mobility of which is rather in an up-and-down direction; and this distinction is of service, inasmuch as the omental and mesenteric growths present in many respects a close resemblance to the dislocated viscus, a condition which is rarely seen in men.

The *prognosis* of these tumours is invariably grave; if left alone they run a course of but a few months after their first recognition; and, unless quite small, their removal is either impossible, or the attempt will probably hasten death. Yet this is not always so; Professor Llobet records a case of complete recovery after removal of a mesenteric sarcoma together with a considerable piece of small intestine to which it was inseparably adherent. A diminution in size has been sometimes observed after a simple exploratory laparotomy.

Carcinoma. A. *Primary.*—The existence of primary cancer of the peritoneum is much in dispute; opinions differ both as to the exact microscopic characters of certain growths, and also as to the precise genetic relationships of the serous membrane. Histologically there can be no doubt that primary tumours, connected with the peritoneum or with the subperitoneal connective tissue, do occur which present wholly, or in parts of them, the appearance of masses of cells epithelial in character, and arranged in alveoli which are formed of interlacing strands of connective tissue—the appearances, that is, of carcinoma. By those who deny that serous tissue can give rise to epithelial growth such neoplasms are named "alveolar sarcoma"; and some even affirm the possibility of distinguishing "a delicate reticulum between the individual cells, a condition never found in cancer" (22, p. 71).

Two views are held of the origin of the lining membrane of the primitive body cavity (cœlom), which later is differentiated into the pleural, pericardial and peritoneal sacs. On the one hand it is regarded

as being developed in the thickness of the mesoblast and primarily continuous with the vascular system, both blood and lymphatic. Inasmuch as it is from the mesoblast that the osseous, cartilaginous, and other of the connective tissues are developed, as well as the muscles (plain and striated), vessels and nerves, it is asserted that no true epithelial growth can originate from the mesoblastic structures, and consequently that a primary carcinoma of the peritoneum is a contradiction in terms. But the mesoblast itself is mainly if not entirely derived from the two fundamental layers of the blastoderm, the epiblast and hypoblast, both of which are the precursors of the epithelial tissues of the adult; and their potentiality for epithelial development, though ordinarily in abeyance, might be retained through the intermediate stage of the mesoblast. Moreover, that some epithelia may arise from the mesoblastic cells is evident from the formation of the Wolffian bodies from which the renal and generative glands are derived. On the other view that the body cavity is an outgrowth from the primitive alimentary canal with the hypoblastic covering of which its lining membrane is continuous, the possibility of a development of true carcinoma in connection with the pleuro-peritoneum is at once obvious; the probability being determined by other conditions. In accordance with this explanation the serous sacs are not at first connected with the lymphatic system, the communications which undoubtedly do exist in the adult being established later by means of the stomata.

However this may be, it is true, nevertheless, that growths believed to be strictly limited to the peritoneum — the serous rather than the subserous layer — and to this extent to be regarded as primary, are occasionally met with. They present the microscopic features of carcinoma or, as already mentioned, are of a mixed character, in which sarcomatous and carcinomatous features are combined. To such growths the name "endothelial sarcoma" is applied by those who hesitate to admit their epithelial nature. A close relation in the arrangement of the alveoli with the lymphatic vessels has been described, the masses of epithelioid cells being derived from a proliferation of the endothelium of those channels or from the covering layer of the serous membrane itself.

B. *Secondary.* — Although carcinomatous affections of the glandular structures of the abdomen are of fairly frequent occurrence, implication of the peritoneum secondary thereto is not so common as might be supposed. It is by extension of the growth from the diseased viscus, as well as by secondary infection by way of the lymphatic vessels, that the serous membrane is implicated, and probably the ovaries are the commonest source; less often the stomach, intestines, liver, gall-bladder, and pancreas, and very rarely the œsophagus or breast. Once the serosa is invaded, as Dr. Coats points out, particles of the tumour may be carried about to different parts of the cavity and, by means of the stomata, into the subserous tissue also; the resulting growths are then really subperitoneal, and covered by an unbroken layer of serous membrane. Secondary

carcinomata faithfully reproduce the microscopic structure of the tumour whence they originated.

Frequency. Age. Sex.—Trustworthy statistics are wanting to enable any definite statements to be made as to the frequency of peritoneal cancer, whether primary or secondary. Almost certainly many of the recorded cases, especially of the former, were really sarcoma. For lack also of precise figures the relative incidence of the disease in the two sexes cannot be positively stated, although from the frequency of cancer of the pelvic reproductive organs it would seem to be commoner among women. As regards age, it is mainly a disease of middle and later middle age, though not unknown in the young.

Morbid anatomy.—The general appearance of growths of this character varies much. In cases which, provisionally at least, are to be regarded as primary, the new growth occurs in a miliary form, in appearance not unlike tubercle. Scattered over the peritoneum, visceral and parietal, and sometimes also invading the pleuræ, are numerous small flattened nodules varying in size from a grain of sand to a pea or even larger: they are sometimes pedunculated and in clusters. As a secondary infection, according to Dr. Fenwick, this form of carcinoma is met with more often as a sequel of cancer of the gall-bladder, pancreas, or ovaries. Dr. Hebb has recorded a case in which it was secondary to a similar condition of the right pleura. Dr. Fenwick has also drawn special attention to the mesentery and mesocolon as being most affected, with the result that they become much puckered and contracted, and drag the intestines backwards towards the spinal column.

In another group of cases the new growth forms large masses, the omentum being the special seat of morbid change, becoming converted into a cord-like tumour, of a variable degree of hardness, lying across the abdomen, below the transverse colon. It is seldom confined to this structure, however, numerous nodules of different size being spread about the peritoneal cavity; often also with scattered miliary formations, as already described. Sometimes this more massive form would seem to be primary. Dr. S. West has published a case of a woman aged fifty-two years, where there were found an irregular flat tumour of the omentum $1\frac{1}{2}$ inch thick and weighing 21 oz., and many much smaller masses distributed over the root of the mesentery; there was none elsewhere in the body. On microscopic examination the growth presented the characters of scirrhous carcinoma.

A third form in which malignant disease of the peritoneum occurs is that denominated "colloid cancer." Concerning the exact nature of this disease, and especially of its mode of origin and relations, much remains to be learned. It forms the largest abdominal tumours with which we are acquainted. Dr. Osler mentions one weighing over 100 lbs. In structure the new growth, which tends to involve the entire peritoneal cavity and its contents, consists of large alveoli bounded by a connective tissue framework, and containing a homogeneous coll id matter with occasional indications of epithelioid cells singly or in groups. Although

the bulk of the material is of this soft gluey character, it fills the containing spaces so tensely as to form tumours of very considerable hardness. So far as present knowledge goes there is no reason to suppose that such growths are primary; they begin rather as carcinoma of some viscus, probably of the stomach or intestine, and extend therefrom to the peritoneum; while the cellular constituent tissues of the growth and the stroma undergo a mucoid or colloid degeneration until spaces large enough to be visible to the naked eye are formed. Whether some or indeed all of the neoplasms included in this group would not be more properly regarded as myxo-sarcoma is as yet uncertain.

The condition of the peritoneum in these cases is noteworthy. When the growth is mainly of a disseminated miliary character the serous membrane is much thickened, sometimes to an enormous extent; partly from a diffuse infiltration of the membrane by carcinoma, and partly from an associated inflammatory new formation. Reference has been already made to the contraction of the mesenteries which may result from this thickening, and to the displacement of the intestines which ensues. Even in the absence of this condition peritoneal adhesions between adjacent viscera, or between them and the parietal layer of the serous membrane, are almost always to be found, with the result of an adhesive peritonitis determined by the new growth. The extent to which they are carried is extremely variable; sometimes they mat the various parts together, especially in and about the pelvic cavity, into inextricable masses. Usually also there is some effusion, a few ounces or many pints; in the miliary form it is very considerable in amount. The fluid presents the ordinary characters of an inflammatory exudation or, as is often the case, it is hæmorrhagic. Sometimes it has been chylous, and a remarkable case of secondary miliary carcinoma of the peritoneum, causing engorgement and rupture of the mesenteric lacteals with chylous ascites, is recorded under the care of Dr. Fenwick. Chylous bullæ were also seen in the intestinal submucosa. The mesenteric and retroperitoneal glands are often unaffected, or only very slightly enlarged.

Clinical characters.—The usual features of malignancy are exhibited in a supreme degree by most cases of peritoneal cancer; since in addition to the evil influence of the growth itself there is usually also a direct interference with the digestive and absorbent organs.

As is commonly the case in abdominal cancer, whatever the organ primarily affected, the onset is vague and insidious—a general sense of failing health and bodily weakness rather than any definite symptoms being the earliest phenomena observed. Moreover, it is practically impossible when the growth is secondary, as it usually is, to discriminate between the manifestations of the primary affection and those due to the peritoneal implication. Neither at the outset nor in the course of the disease is pain by any means a constant or often a serious symptom. Occasionally attacks of griping and colic, often accompanied by flatulence, may be succeeded by considerable intervals of complete freedom from more than a little abdominal discomfort. But, on the other hand, in

some cases pain is a prominent and almost a constant symptom throughout; and the exact reason for the differences in this respect remains to be found. Tenderness also is variable in extent, but sooner or later it usually appears. The temperature, which inclines to be subnormal, may exhibit exacerbations which are attributed to a slight spread of peritonitis, especially if they occur after tapping the abdomen. All degrees of digestive disturbance are observed; the appetite is capricious or wanting, and even at first is rarely well maintained. Constipation is the rule and, determined as it is by obstructions of the intestine from the pressure of tumours and fluid, from stricture of its lumen by new growth in its walls, or from twistings and displacements due to contraction of the mesentery, is often most obstinate. Later in the progress of the case an intractable diarrhœa may set in and hasten the fatal end; or attacks of diarrhœa may alternate with constipation. Vomiting is sometimes met with. The malnutrition induced by the deficient nourishment added to the specific effect of the growth induces a weakness and fatigue marked from the first, and it is this condition, indeed, which may be the first to attract attention to the real state of the patient. Soon it is accompanied with an increasing loss of weight, anæmia, and the invasion of a characteristic cachexia, symptoms which steadily progress.

The *physical signs* will necessarily vary with the size and situation of the growth, the presence or absence of a visceral tumour, the extent of the ascites, and the pressure effects to which these conditions give rise. When the new formations are entirely of a miliary character they cannot be detected, and their presence can only be inferred; and, since in such cases there is extreme ascites, the nature of the malady may be overlooked. When the nodules attain an appreciable size they may be felt with varying distinctness through the emaciated abdominal wall, those more especially which have formed on the parietal peritoneum or are adherent thereto. The facility with which the abdominal contents can be palpated will necessarily depend in great measure on the amount of the ascites. The omentum, which is rarely free from the new growth, often forms the largest and best recognised tumour; it is thickened and rounded in shape, sometimes partly broken into two or more masses, or flattened and moulded to the curve of the anterior wall of the belly. The greatest difference exists as to the range of mobility of the tumours, but their tendency is to become fixed by peritoneal adhesions. Unless the distension from fluid be extreme, when a general dull note will be obtained by percussion, the resonance over the abdomen is irregularly distributed in accordance with the size and situation of the tumour or tumours. Dr. Fenwick has particularly drawn attention to the dulness along the course of the colon in those cases where the intestines are retracted to the spine by a thickened and shrunken mesentery; and not only are the flanks then dull, but, on placing the patient in the genu-pectoral position, the usually resonant area below and to the outer side of the kidneys is dull on one side or on both. Considerable irregularity in the contour of the surface of the abdomen is rendered more apparent by the wasted

integuments. A characteristic appearance is that presented by the umbilicus, the skin around it being sometimes red and much thickened. The inguinal glands are frequently enlarged.

The duration of the disease after the existence of the growth has been recognised rarely exceeds six months, and sometimes it is much shorter; the miliary variety is especially acute in its course.

Diagnosis.—Before the peritoneal tumours can be detected by physical examination a diagnosis of malignant disease cannot be made, though it may be suspected; especially if a visceral growth be known to exist.

The miliary form with its considerable ascites and absence of palpable tumour may be mistaken for hepatic cirrhosis, but the history of the case and the habits of the patient must be considered. In the latter affection, also, the abdominal distension is usually greater and the enlargement of the superficial veins much more marked, owing to the direct interference with the venous return.

The distinction between peritoneal cancer and tuberculous peritonitis is often hard to make. There are the same fulness of the abdomen, moderate ascites, the very slight mobility of the fluid which is circumscribed by the presence of adhesions, an omental tumour, and, very commonly also, palpable nodules distributed over the abdomen. The removal of the fluid does not facilitate the diagnosis which must depend upon other circumstances, such as the age—the tuberculous disease being much more frequent in children and young persons; the presence of tubercle in other organs; the steady progress, and, as a rule, shorter duration of the malignant disease—the tuberculous affection being often marked by periods of quiescence or even of partial improvement. A discharge of pus from the umbilicus is strongly in favour of tuberculous peritonitis; on the other hand, red nodular swellings in this locality suggest carcinoma. But with every care and by skilled observers the disease may remain doubtful.

The *prognosis* is invariably bad, the peritoneal affection frequently contributing to the fatal progress of the primary disease. Operative treatment, beyond paracentesis for the relief of fluid distension, is to be deprecated; the removal of the multiple growth is impossible, and the attempt only hastens the inevitable end. Anodyne applications, a judicious use of morphia internally, due attention to the bowels, and the administration of the most digestible and nutritious diet are the sole means of treatment at our disposal.

Angioma.—An unique case of nævus of the peritoneum has been recorded by Mr. Arbuthnot Lane. A male child, aged seven years, came under his care suffering from a tumour which had been noticed from birth, since which time it had increased in size until it came to form a prominent swelling occupying the right side of the abdomen. It extended from near the middle line in front to the loin posteriorly, and from the margin of the thorax above to the iliac crest; it appeared to involve the abdominal wall, and fluctuation was felt over the most prominent portions

In the course of a successful excision of the growth, several large cysts, some nearly as large as an orange, and many smaller ones, were opened and found to contain liquid blood, their inner aspect being smooth and polished. The tumour appeared to start in the outer surface of the peritoneum, forming livid projections into the peritoneal cavity; and externally it invaded the muscles of the abdominal wall. It extended upwards beneath the diaphragm, and backwards into relation with the kidney; it proved to be nævoid in character, and was apparently undergoing rapid degeneration.

Under the name of plexiform angio-sarcoma Waldeyer has described certain large tumours, connected with the peritoneum, consisting of new-formed blood-vessels, the outer coats of which have become the seat of extensive colloid change.

A point of special interest in these vascular growths is their possible relation to the sarcomata, for by some pathologists the latter are said to be derived from angioblastic tissue (19).

Cysts.—Reference has been made to the liability of certain growths, especially the sarcomata, to undergo cystic change; and this may sometimes be so extreme as to give rise to a tumour which is mainly composed of cysts, the undegenerate hard parts forming but a relatively small portion of the mass. Besides these, however, cysts of various kinds are occasionally to be found in connection with the peritoneum or its subjacent connective tissue; of some of these the precise nature and mode of origin are obscure, though others are sufficiently definite in character.

Hydatid cysts.—The best-defined and probably on the whole the most common cysts found in connection with the peritoneum are those of the echinococcus. The late Dr. Davies Thomas, out of 1897 cases of hydatid disease found the peritoneum and subperitoneal tissue affected in 26; that is, in about 1·37 per cent. Hydatid cysts are very seldom confined to these parts, almost always some of the viscera or other structures are involved also.

In the majority of cases the cysts are multiple, and may number a few or hundreds, varying in size from a pin's head to a cricket-ball or larger. These small-sized cysts may be either sessile or pedunculated. Sometimes the tumour is single; it may then attain a very considerable size, or one or perhaps two large cysts may completely fill the abdomen in such a manner as to simulate ascites. Dr. Fenwick (6) has noted that in such cases the cyst wall is adherent to the parietes, which is not usual when the cysts are smaller; and this circumstance contributes to the resemblance which the physical signs have to peritoneal dropsy. Suppuration is sometimes set up within these tumours, or the contents may be converted into a putty-like mass.

Probably most of these growths begin in the subserous tissue, either retroperitoneal, or between the layers of the omenta, mesentery, or broad ligament; or beneath the peritoneal covering of the viscera such as the liver and spleen, the surface of which they indent. When

developed upon the peritoneum they are most probably due to direct infection from a ruptured or punctured cyst, and in such cases are likely to be excessively numerous. As a rule the ectocyst tends to be thicker in peritoneal hydatids than it is in similar tumours elsewhere. [*Vide* art. "Hydatid Disease," vol. ii. p. 1102.]

Dermoid cysts.—Cysts of this class, with their characteristic contents of fatty matter and hairs enclosed in an epithelial lined structure homologous with skin, are sometimes met with attached to the peritoneal membrane and growing into the cavity, or in the retroperitoneal tissue. The latter are probably the more frequent, and are developed in patients of both sexes behind the rectum; sometimes they grow to a great size and extend upwards behind the serous membrane as high as the umbilicus, causing considerable displacement of the adjacent viscera. As Mr. Bland Sutton points out, those met with in this situation may be dentigerous. The intraperitoneal dermoids may be very numerous, the result of secondary infection of the membrane from rupture into the serous sac of an ovarian cyst of the same character. Such tumours rarely attain a large size, and "may take the form of minute granules on the peritoneum, each of which is furnished with a tuft of delicate lanugo-like hair, or may hang from the under surface of the liver, or form clusters like cherries upon a branch, or be embedded in adhesions between coils of intestine." (22). Such growths are exceedingly rare. Very large single cysts of this variety have also been met with containing several pounds of sebaceous matter with hair. Some of these have doubtless originated behind the peritoneum, and extended between the layers of the mesentery; but others have been found attached to the omentum, and Mr. Alban Doran is strongly of opinion that the latter are really ovarian in origin, "but have become adherent to other structures and then separated from their pedicles" (6).

Like dermoid cysts generally these tumours are very uncertain and irregular in their growth, and after years of quiescence they may increase rapidly in size. Owing to contamination from the intestine, to which they may become adherent, they are liable to suppurate; and may then burst into the bowel, the vagina, or on the surface at the umbilicus.

Cysts of doubtful nature.—Scattered throughout medical writings are many records of cysts connected with the mesenteries, omenta, peritoneum, or subperitoneal tissue; not sufficiently numerous, however, nor described with sufficient detail to permit of exact classification or of complete recognition of their character and origin. They are frequently designated as "hydatid," a name which formerly had a wider meaning than its present restricted application, and this tends to confusion; but it is also probable that some were really sterile echinococcus cysts; these should be detected by the laminated character of the wall and the peculiar nature of the confined fluid, even in the absence of scolices and hooklets (*vide* art. "Hydatid Disease," vol. ii. p. 1120).

Other cysts of this indeterminate group probably originate from obstructed and dilated lymphatics. Such are usually met with in the

substance of the omentum (the other tissues of which having more or less disappeared), where they form pedunculated clusters of ovoid or pyriform bodies resembling bunches of grapes; or they may appear as elongated cylindrical swellings several inches in length and of the diameter of the finger. They are lined with a layer of columnar epithelium, and contain a clear, yellowish, slightly viscid albuminous fluid. Closely similar to these, if not identical with them, are the mesenteric cysts which contain chyle. Considerable effusions of chylous fluid have been met with between the layers of the mesentery or in the retroperitoneal tissue, due probably to rupture of one or more chyliferous vessels, which form large swellings containing several pints of fluid (20). Such tumours, however, are not true cysts, since the fluid is not contained in a very definite membranous sac, but within the accidental boundaries of the surrounding tissue. Similarly, effusions of blood (hæmatoma), the result of injury, may be found in the same situations.

Some of the encysted collections of serous fluid which occur in the peritoneal cavity, and also (as there is good reason to suppose) in the sac of the great omentum, are inflammatory in origin and are really due to localised peritonitis, the fluid being confined by the formation of adhesions.

Exceedingly thin-walled cysts, of rare occurrence in the omentum and also in the subserous tissue, are described, containing sometimes several pints of clear serous fluid. The wall itself consists of a delicate connective tissue, without any evidence of an epithelial lining. No adequate explanation has been given of the nature of these cysts.

Lastly may be mentioned the so-called "sanguineous peritoneal cysts," which are interesting from their possible relation to pancreatic cysts. More than twenty of these cases have been collected and compared by Dr. T. Fisher, but some would appear to have been inflammatory effusions into the lesser peritoneal cavity following on injury to the pancreas (see "Cysts of the Pancreas," in the 4th vol. of this System).

Clinical characters of peritoneal cysts.—The signs and symptoms of these abdominal swellings much depend upon their size and their situation. Inasmuch as they tend to interfere but little with the general nutrition, their existence is often unsuspected, and is revealed accidentally during life, or unexpectedly after death. It may be said generally that the symptoms to which they give rise are in the main due to the pressure they exert on various parts; yet even these often amount to little more than a vague sense of discomfort and weight. Some interference with the intestinal peristalsis may be induced, even to the extent of moderate obstruction, and vomiting sometimes occurs; or micturition may be rendered difficult, or too frequent, from the pressure of the tumour on the bladder. But it is seldom that a cyst, however large, leads to any ascites from compression of the portal vein, though this may follow when it is situated in the small omentum, a position in which it may also cause jaundice by obstructing the common bile-duct. When the abdominal cavity is greatly filled by many hydatid cysts, the diaphragm may be so pushed upwards as seriously to embarrass the action of the heart and

lungs. Not often is pain, or even tenderness, complained of; and the presence of these tumours rarely leads to peritonitis, unless evacuation of their contents into the peritoneal cavity should occur; and not always then. The occurrence of suppuration in a cyst is usually accompanied by the general symptoms of that condition—fever, rigors, and sweating—to a greater or less extent.

Great variability necessarily exists in the *physical signs* of tumours of this class, according to their size, their situation, and their mobility. The last characteristic is best marked in the smaller and medium-sized cysts attached to the omentum or mesenteries, and it may then be manifested to a considerable degree. The existence of adhesions will obviously limit the range within which the swellings may be moved, and the cysts which are retroperitoneal tend to be quite fixed. As a rule it is rather in a side-to-side direction than from above and downward that the mobility is greatest. The feel of the tumour is tense and elastic, but it is not always easy to distinguish a cyst from a solid growth, especially if it be deeply situated. Fluctuation is perceptible when the growth is large, and the characteristic "fremitus" may be elicited when a hydatid is near to the surface. The cyst may appear smooth and uniform, or nodular and irregular, the latter being generally the case with hydatid tumours.

The *duration* of these various forms of cysts is most uncertain and difficult to ascertain. The time of their onset can scarcely ever be known; but as contrasted with most of the solid tumours of this region, the cystic swellings are of far longer duration, and indeed may remain with little or no change for years. They are, however, liable to suppurate, and then to give rise to more active and serious symptoms; or they may burst either into the peritoneal or pleural cavities and set up grave mischief of a secondary character.

The *diagnosis* of these cysts is often very difficult. Those which are deeply situated behind the peritoneum may be easily mistaken for solid growths in that situation, the fluid character of the contents not being recognisable, even if there be no undue thickness of the abdominal walls; those which originate in the pelvis, or dip down into it, may be excusably confounded with cysts of the ovary or its appendages. When, however, the tumour is single, and its cystic characters are recognised, it may still be impossible to assert that it is hydatid in nature. It is commonly recommended to puncture the swelling and to examine the fluid withdrawn; but apart from the fact that a procedure apparently so simple is not wholly unattended with danger, grave symptoms and even death having followed from an exploratory tapping, it is, in my opinion, better to remove the tumour at once; it is always liable to increase in size and to become troublesome, and, when free from adhesions, the operation is accompanied with but a minimum of risk.

W. H. Allchin.

REFERENCES

1. ADAMI, J. G. "Retro-peritoneal and Perirenal Lipomata," *Montreal Med. Jour.* Jan. and Feb. 1897.—2. *Ibid.* Feb. 1894, p. 628. "Mixed Carcinoma and Sarcoma of Peritoneum."—3. ANDERSON, W. "The Surgery of the Subperitoneal Tissue," *Brit. Med. Jour.* 1896, Oct. 17.—4. CIMA, F. *Pediatria*, May 1896.—5. COATS, J. *Manual of Pathology*, 3rd ed.—6. DORAN, A. "Notes on so-called Non-ovarian Dermoid Abdominal Tumours," *Roy. Med. Chir. Soc. Lond. Trans.* 1885, p. 235.—7. FENWICK, S. *Lectures on Obscure Diseases of the Abdomen*, 1889.—8. *Idem. Brit. Med. Jour.* 1891, ii. p. 598.—9. FISHER, T. "Peritoneal Sanguineous Cysts," *Guy's Hosp. Rep.* xlix. 1892.—10. HEBB, R. G. "Primary Cancer of Pleura," *Path. Soc. Lond. Trans.* 1893, xliv. p. 5.—11. HOMANS, S. "Two Cases of Removal of Enormous Fatty Tumours by Abdominal Section," *Lancet*, 1883, i. p. 449.—12. LANE, W. ARBUTHNOT. "Case of Extensive Ulcers of Peritoneum," *Clin. Soc. Lond. Trans.* 1893, xxvi. p. 5.—13. LAUWERS. *Ann. de Med. Belge*, 1891, p. 311.—14. LEBERT. *Abhandlung der prakt. Chirur.* 1848.—15. LLOBET. "Ablation d'un sarcome du mesentère," *Rev. de Chirur.* 1891, p. 676.—16. LOCKWOOD, C. B. "The Diagnosis of Retro-peritoneal Sarcoma," *Med. Soc. Lond. Trans.* 1895, p. 324.—17. MORTON, C. A. "Case of Sarcoma of Peritoneum," *Path. Soc. Lond. Trans.* 1893, xliv. p. 82.—18. PICK, T. P. "Enormous Fatty Tumour of Abdomen," *Path. Soc. Lond. Trans.* 1869, xx. p. 337.—19. PILLIET. "Angiome de la sous narine," *Bull. de la Soc. Anat.* 1894, p. 621.—20. RASCH, A. A. F. "Large Chylous Cyst of the Mesentery," *Obstet. Soc. Lond. Trans.* 1889.—21. SHIELD, A. M. "A Case of a Large Solid Tumour removed with Success from the Retro-peritoneal Space," *Roy. Med.-Chir. Soc. Lond. Trans.* 1897.—22. SUTTON, J. BLAND. *On Tumours*, 1894.—23. THOMAS, J. DAVIES. *Hydatid Disease*, 1894.—24. TREVES, F. "A Case of Lipoma of the Broad Ligament," *Clin. Soc. Trans.* 1893, vol. xxvi. p. 101.—25. *Idem.* "A Review of the Surgery of the Peritoneum," *Brit. Med. Jour.* 1896, Oct. 31.—26. WALDEYER. "Die Entwickelung der Carcinome," *Virchow's Archiv*, Bd. lv. p. 134.—27. WEST, S. "Primary Scirrhous Cancer of the Omentum," *Path. Soc. Lond. Trans.* 1890, xli. p. 95.

W. H. A.

DISEASES OF THE BOWELS

PHYSIOLOGY OF FÆCAL EVACUATION

In order to understand clearly the action of the various conditions which produce constipation and diarrhœa, it is necessary to consider the physiology of the fæcal evacuation. The intestinal contents are moved onwards by peristaltic action in a direction from the stomach towards the anus, until they arrive at the rectum, where their presence, by reflex action, calls upon the muscles of the abdominal wall to aid in their expulsion.

The intestinal walls consist of an external peritoneal covering, under which lies a thin longitudinal and a thicker circular muscular coat; below this is a loose layer of connective tissue, and internally lies the mucous membrane. The muscular fibres in the small intestine are nearly equally distributed around the whole of its walls; but in the large intestine they are thin over the greater part of the circumference of the bowel, and are collected chiefly into three bands nearly equidistant from each other. These bands are shorter than the bowel itself, so that they give the bowel the appearance of being drawn into a number of saccuii. The arrangements both for secretion and motion are contained in the bowel itself. This is shown by the facts that after their entire separation from the body the intestines continue to exhibit vigorous peristaltic action; and that, when all the nerves passing to the small intestines are cut, the mucous membrane of the small intestine secretes copiously.

The movements of the intestine are of two kinds: (i.) Peristaltic, in which contraction takes place at some part of the bowel, and gradually passes down for a certain distance; (ii.) swinging movements.

It is very rare for peristaltic movement to continue along the whole bowel; it usually extends for a certain distance only, and then stops; but a new movement may begin shortly afterwards at the place where the first peristaltic action stopped, so that by successive movements of different parts of the bowel the contents are moved on from the one end to the other. The swinging or pendulum-like movements are probably produced chiefly by the longitudinal fibres. When observed in the intestines after the abdomen has been opened, they do not seem to have any particular effect one way or another; but in all probability they are useful in restoring the bowels to their former position after they have shifted from it by peristaltic action.

Peristaltic action is usually moderate; but it may sometimes be exceedingly violent, both in amount and duration. Occasionally, violent peristaltic action occurring over short distances, but not extending down the bowel, appears simply to give rise to borborygmi; but, when excessive, and extending along the whole length of the intestine, it gives rise to diarrhœa. The ordinary peristaltic action and the swinging movements may be regarded as the normal movements of the bowel. The excessively violent peristaltic action which gives rise to diarrhœa may be looked upon as pathological. A common form of pathological movement is a tonic contraction, which converts the soft and elastic bowel almost into a rigid rod. Not infrequently this can be felt through the abdominal wall in persons suffering from chronic intestinal catarrh, from obstruction, or from abnormality in the nerve-supply, as in lead colic; and when it occurs extensively, as in lead colic or certain diseases or abnormal conditions of the brain, the abdomen becomes retracted, and assumes the form to which the name "scaphoid" has been applied. The short peristaltic actions which give rise to borborygmi may be looked upon as intermediate between pathological and physiological conditions, inasmuch as they do not occur under ordinary circumstances, but yet cause no pain or other abnormal symptom. They are so commonly due to emotional causes that their occurrence was formerly regarded as an evidence of the emotion of compassion; and, indeed, the term "soundings of the bowels" was used by the old Hebrew prophets, for example, as a synonym for compassion itself: "Where are the soundings of thy bowels towards me?" or again, "My bowels shall sound like a harp for Moab."

It has been supposed by some authors that in peristaltic action the stimulus to contraction is simply passed on from one muscular fibre to another; but this seems unlikely, and it is much more probable that, although the stimulus may travel to some extent along the muscles, it does so chiefly along the nerves. The movements of the bowels originate in nervous impulses supplied to the muscular fibres by the nervous plexus of cells and fibres, usually known by the name of Auerbach's plexus, which lies between the longitudinal and circular layers of the intestinal muscle. The small intestine, and the greater part of the large, have their movements regulated from the spinal cord and sympathetic plexuses. As the greater part of the intestine works independently of the will, the nervous fibres which reach it from the cord pass through the gangliated chain of the sympathetic as well as through plexuses in the abdomen itself. The stomach and rectum, however, are more under the control of the will, and they are partly innervated directly from the brain and spinal cord. The stomach receives its innervation in part directly from the brain through the left vagus, and in part indirectly through the right vagus which enters the solar plexuses. The lower parts of the sigmoid flexure and the rectum receive fibres from the first, second, and third sacral nerves, which do not pass through the gangliated sympathetic cord, but enter into the hypogastric plexus and then pass onwards to the intestine.

The secretion of the smaller intestine has its regulating centres in the superior and inferior mesenteric plexuses; for if all other parts of the nervous system be destroyed while these are left intact, no abnormal secretion occurs; but if they are destroyed a copious secretion takes place in the small intestine, just as if all the nerves which pass through it had been cut.

The vagus and the splanchnic nerves which enter into the solar plexus have different functions. The vagus, as a rule, stimulates intestinal movements, while irritation of the splanchnics arrests them; but, under certain conditions, these effects may be reversed, and the vagus may have an inhibitory and the splanchnic a stimulating action. It has been stated by von Basch that the vagus innervates the longitudinal muscles of the intestine, and the splanchnic the circular muscles. The splanchnics also contain vaso-motor fibres which cause the intestinal vessels to contract, and, through the anæmia which they can thus produce, may exert a secondary action upon the intestine. Both the vagi and the splanchnics are afferent as well as efferent nerves; for whereas the vagi probably may convey a sensation of pain, it is certain that the splanchnics do so when strongly stimulated.

From the description just given of the nervous connexions of the intestine, and of the different results which may be produced by stimulation of the nerves under different conditions, it is obvious that stimulation of the mucous membrane, either mechanically or chemically, may give rise to very different results at different times. At one time it may cause increased secretion, at another dryness of the bowel; at one time increased peristalsis with diarrhœa, at another diminished peristalsis and constipation. A moderate amount of distension, either by fæces or gas, usually increases the peristaltic movement; excessive distension, on the other hand, tends to their abolition.

The emotion of compassion, as already said, usually tends to produce limited peristalsis with borborygmi; the emotion of fear to cause general peristalsis with evacuation of the bowels. In some persons emotions of almost any kind, even pleasurable, have a similar action; but depressing emotions, such as anxiety and grief, frequently appear not only to cause dryness of the tongue, but also dryness of the intestine, sluggish peristaltic movement, and great constipation. Excessive stimulation, either of the inhibitory or motor nerves of the intestine, appears to result in a reaction, during which fibres of the opposite kind have the preponderance; so that diarrhœa is apt to be followed by constipation, and conversely. This result may ensue whether the diarrhœa or constipation be caused by the administration of purgatives and astringents, or be brought about by wholly different causes. It is evident, also, that if the opposing nerves, either for secretion or for motion, should be nearly exactly balanced in their excitability and power, no irritant applied to the intestine would have any result, however strong it might be; and consequently most powerful purgatives, such as jalap, elaterium, or croton-oil, might be given without producing purgation.

Constipation

Constipation is a tendency to the retention of fæcal matters in the intestine; the evacuations either being too small or occurring too seldom. Constipation is one of the commonest troubles of civilised life; and it occurs so frequently in people who are otherwise perfectly healthy, that to a great extent it must be regarded not as a disease, but as the natural result of artificial conditions acting upon a healthy body. The natural stimulus to the movement of the bowels is afforded by the mechanical or chemical irritation exerted by the indigestible residues of food. In savage life these are abundant, because the food is not only coarse in quality, but imperfectly cooked. In civilised life the indigestible parts are to a great extent removed from food; and even those that remain are so much softened by cooking that they do not exert the same irritating action upon the bowel that they would otherwise do. Thus in primitive communities the grain of all kinds, which forms such an important element in food, is bruised or pounded; and the meal, with a liberal admixture of sand and dust, is made into cakes, or boiled without further preparation. But in highly civilised societies the case is very different, for a great deal of the outer part of the wheat or other grain is removed, and the fine starch contained in the interior is the only part employed in making bread. This fine starch is wholly digested in the intestine, leaving little or no residue to stimulate the bowels; whereas the exterior of the grain, consisting as it does of hard cellulose, is almost indigestible, and passes through the whole intestine with very little change. It frequently happens that when people no longer restrict themselves to bread made with fine flour, and return to the more primitive bread, made either from whole meal or from bran, the bowels which had previously been constipated become regular. Many of the dietetic methods of treating constipation consist in giving food which contains cellulose in a more or less hard and indigestible form. Thus patients are encouraged to eat bread made of whole meal or bran; apples stewed or raw; figs, raw or in puddings; prunes or marmalade. Marmalade partly consists of the hard skin of the orange, which is cut into strips, and being very sparingly digestible exerts a mechanical stimulating effect upon the bowels; in addition to the mechanical effect, however, there is a physical, perhaps we might also say a chemical action exerted by sugars and neutral salts of the alkalies. Both of these are constituents of fruit, and the neutral salts without the sugar occur in other vegetables. The sugar exists in large quantities in such fruits as figs, prunes, tamarinds, and the like; and sugar, given alone in the form of honey, golden syrup, or still better the old-fashioned black treacle, tends to act as a laxative. It is, perhaps, still more efficient when given as marmalade or jam, because the action of the sugar is combined in these substances with the mechanical stimulation contained in the skins or seeds of the fruit. Vegetables, such

as cabbage, spinach, sprouts and salad, have a double stimulant action, in part mechanical from their indigestible constituents, and in part chemical from the neutral salts they contain. Moreover, some of them increase the movements of the bowels by giving rise to the evolution of a certain quantity of gas which aids their expulsive action.

The causes of constipation are very numerous, but they may be ranged under two main heads: firstly, lessened expulsive power; secondly, increased resistance. The diminished expulsive power may be (i.) in the peristaltic action of the intestine; (ii.) in the action of the diaphragm and abdominal muscles which aid the intestine in the expulsion of its contents.

The diminution of expulsive power in the intestine itself is rarely due to alteration in the muscular fibres. In some cases it would appear that the muscular coat of the intestine is naturally weak, although the voluntary muscles in the same person may be well developed; in some men, likewise, the heart is small and out of proportion to the size of the body. The intestinal muscles, again, like the heart, may be weakened or degenerated by prolonged disease, as by fevers; but the intestines seem to have a greater resisting power than the heart to a high temperature, and do not show degeneration to the same extent as a consequence of prolonged febrile disease. Even in severe and prolonged diseases, when the voluntary muscles have become extremely atrophied, the intestinal muscular walls show little alteration. Lessening of the expulsive power of the intestine is, therefore, chiefly due to alteration in its nervous supply. Occasionally this may be of central origin, as in the extremely obstinate constipation of melancholia, mania, and other cerebral diseases; but it frequently appears to be of reflex origin, or due to some peripheral condition in the intestinal nerves themselves.

In chronic catarrh of the intestine there is a tendency to constipation, which may be due to weakening of the muscular fibres of the intestine, as well as to the nerves; in peritonitis it is probable that both agents are affected. As a rule, however, sluggishness of the intestinal movements depends rather upon the want of stimulation or the inactivity of the nervous system than upon the condition of the muscular fibres. One powerful cause of constipation is undoubtedly the presence of lime salts in drinking-water. The mode of operation of these salts has not been ascertained, but it is quite possible that it is due to an action of the lime upon the muscular fibre, rendering its contraction slower than before; without doubt lime has this action upon involuntary muscular fibre generally.

Diminution in the expulsive power of the abdominal muscles may be due to great distension, such as occurs during pregnancy, and as a consequence of repeated pregnancies; to great accumulation of omental fat; or to tumours in the abdomen, such as large ovarian cysts. Weakness of these muscles may also be due to the innutrition consequent upon insufficient exercise. This may occur, for instance, in heart disease, when the patient is prevented by the state of his circulation from taking

exercise; or it may be due to sedentary habits, or due to lack of time, opportunity, or disposition to take exercise. Again, the muscles forming the floor of the pelvis may be lax and weak, and may offer too little of the resistance which turns the fæcal mass from the direction in which it passes into the pelvis forwards to the anus. There may be inability also to bring the muscles of the abdomen into full play on account of cardiac disease, which prevents a patient from straining, or from piles, fissure of the anus, fistula, or other disease in the pelvic organs; in which cases pain during defæcation makes the patient afraid to bear down.

The natural stimulants to the intestines may be either of a mechanical or a chemical nature. The mechanical stimulants are the solid residues of food, or the distension by gases inside the intestine; while those from without are the mechanical pressure to which the intestines are subjected by the diaphragm and abdominal walls during the actions of respiration, locomotion, and exercise of various sorts. The chemical stimuli are certain gases—such as carbonic acid, sulphuretted hydrogen, and marsh gas, sugars, neutral salts, and possibly other substances formed by the decomposition of food by bacteria within the intestines. When the amount of food which enters the intestine from the stomach is small in quantity, or very soft and easily digestible, it naturally exercises a much less powerful stimulus upon the intestine than if it were abundant and contained a good deal of hard residue. The food may be scanty either from famine, from lack of appetite, from irritability of the stomach causing vomiting, or from ulceration and contraction of the pylorus delaying the food in the stomach. The food may be soft and readily digestible in the poorer classes, for whom bread, tea, and farinaceous food with a little sugar are the chief articles of diet; or, in the richer, from the tenderness of the food and good cooking. When the food is not only soft, but contains no indigestible materials, there will be very little residue in the large intestine; and there will be, therefore, very little stimulus to its peristaltic action. When food is too bulky and too indigestible, it is apt at first to give rise to diarrhœa, which is not unlikely to be followed by a reaction; so that those who are living constantly upon poor and sparingly digestible food may suffer from constipation as well as those in whom it is too soft or too well cooked. A similar reaction follows the use of purgatives; so that those who are accustomed to use purgatives in excess of what is required by nature are very apt to suffer from constipation. One very common cause of constipation is the nervous torpor due to habit. Habits in most persons are very easily acquired. One person, if awakened at an early hour on one morning to catch a train, will go on waking needlessly at the same hour for many mornings to come; another person who, disregarding the summons to rise, once deliberately turns over and goes to sleep after his alarm has sounded, is very apt to do so involuntarily and to oversleep himself on subsequent mornings; although the alarm may sound as usual. Thus the nervous system may be trained to excite an action of the bowels at a certain time, so that an evacuation becomes regular; but habitual disregard of the natural call

to evacuate soon leads to a want of sensitiveness to it, and to constipation as the result.

A short time ago I heard of a useful plan employed by a schoolmaster. He sent all the boys under his care in batches to the closets immediately after breakfast, each batch having to keep time to the minute. If any boy failed to have an evacuation in the time allowed, he was bound to report himself to the master in order to allow of proper means being employed to relax the bowels. If this plan were followed at all schools a useful habit would be established, and in many cases a good deal of discomfort might be prevented in after-life.

Dr. Cash has shown that the introduction of food into the stomach acts as a stimulant to movements of the bowels, and the natural time for evacuation in most people seems to be after breakfast. The stimulation of the food in the morning after the long rest during the night leads to the passage into the rectum of the fæcal matter which has already accumulated in the sigmoid flexure; and thus a desire to defæcate is set up. Such calls are not infrequently disregarded, especially by women, from a misplaced sense of modesty; and not infrequently by men who, in their haste to business, either do not go to the closet at all, or cut the act too short. In some cases long railway journeys, or other incidents, may interfere with the daily evacuation; but whatever the cause, the result is apt to be the same. Preoccupation of mind by thought or emotion may also interfere with evacuation; and, as I have already said, cases of nervous disturbance, such as melancholia or neurasthenia, may be associated with most obstinate constipation. The natural evacuation may also be arrested, sometimes voluntarily, sometimes involuntarily, by the dread of pain; or by actual pain from piles or fissure of the anus, or by inflammation in or around the ovary or some other pelvic organ. It is stated that venous stasis, especially in the portal district, is liable to cause constipation; and that, consequently, cases of cardiac or hepatic disease, which lead to portal stasis, are apt to interfere with evacuation of the bowels. This, however, is by no means invariable, and much venous congestion of the bowels may occur without the peristaltic action being lessened. It is, therefore, probable that constipation due to these causes is not directly due to venous congestion of the bowel itself, but rather to some other factor associated with it.

Increased resistance may occur (*a*) from the altered condition of the fæces—dryness, hardness, or bulkiness; (*b*) from resistance to the movement of the intestine from adhesions; (*c*) to diminution in its lumen by fibrous bands running around it, stricture of the intestine itself or of the anal orifice due to old scars, to piles, or to malignant disease; (*d*) to pressure by other parts, such as a retroverted uterus, fibroid or other tumours of the uterus, or new growths in the pelvic walls, uterus, bladder, or prostate.

Increased resistance to the passage of the fæcal mass along the intestinal canal may arise from dryness of the fæces. There are many causes which may give rise to this defect. Too little liquid may be

habitually drunk; either because the patient is not thirsty, or habitually takes little liquid in order to avoid the necessity of evacuating the bladder under circumstances when it might be inconvenient. Others, again, instead of taking water, take wine; and thus, to a certain extent, they cheat the natural desire for fluid. Excessive excretion by the kidneys may lead to dryness of the fæces and constipation; as, for example, in cases of diabetes, where the sugar acts as a diuretic; or where food is taken containing a sufficient amount of neutral salts to act as a diuretic, but not enough to stimulate the intestine. Much exercise, leading to profuse perspiration, may also draw off water through the skin, and dry the contents of the bowels. If the fæcal masses remain in the intestine they tend to become drier and drier from absorption of the liquid they contain; so that if a person, otherwise in health, voluntarily disregards the call to defæcate, and puts off the act for twenty-four hours longer, his motions are usually more scanty, darker, drier, and harder than they would have been if evacuated at the proper time. These hard, dry masses accumulate and tend to obstruct the bowel, and thus further increase the difficulty of evacuation. Thus if a healthy man is obliged to pass over a day without evacuating the bowels, the movement on the succeeding day is likely to be not only drier and harder than usual, but frequently more scanty. At first this seems extraordinary, because one would imagine that the accumulations of two days would be much more bulky; but the absorption being greater there is less instead of more residue to evacuate. If a longer time passes without evacuation, the hardened fæcal masses become pressed against one another, and the motions which accumulate in the intestine become very bulky and very hard.

Milk sometimes gives rise to exceedingly hard motions. When taken in small draughts at a time, and especially when broken up by admixture with an alkaline water, or by taking a little piece of biscuit between each sip, the milk usually falls in coagula in the stomach, which are quickly digested; but if the milk be drunk in large draughts, it may form large firm curds which, instead of being properly digested, form stringy masses of casein; these become so felted together as to form an exceedingly hard mass which is very difficult to evacuate.

Another cause of dry and hardened fæces is peculiarity in the quality of the water drunk. People living on a chalky soil and drinking hard water are very apt, as I have said, to suffer from constipation. Where this is the case, the patient should drink some of the ordinary bottled waters, such as soda water, potash water, or other aerated water. Not only so, but their tea should also be made with these waters, instead of with the ordinary chalky water; as the quantity of lime in the water taken as tea is sufficient, in some people, if the water be very chalky, to keep up the constipation. It is very difficult for patients supplied with hard water to get their food cooked with water free from lime it is better, therefore, if possible, to secure soft water for cooking purposes.

Exercise has much to do with the movements of the bowels. Want

of exercise tends to cause constipation; partly, I believe, because the bowels do not get a mechanical stimulant applied to them by the contraction of the abdominal muscles. The kind of exercise taken, however, is of no little importance. In ordinary walking on the flat the bowels get little or no mechanical stimulus, because almost the whole of the exertion is thrown upon the muscles of the leg and back; but on walking up a hill, and in many of the so-called Swedish movements, the intestines undergo an alternate compression and relaxation which stimulates them powerfully. In walking uphill, for example, the intestines are squeezed between the diaphragm and the abdominal muscles, both of which parts are called into unusual action by that kind of exercise.

To one class of patients I think exercise is disadvantageous. Not infrequently delicate women, especially those who have some sort of ovarian or uterine irritation, tell you that the more exercise they take, particularly walking exercise, the more obstinate the constipation becomes: whereas when they lie quiet they are perfectly well. In such cases, I believe there is a reflex inhibition of the peristaltic movements of the bowels from the irritated ovaries or uterus; and some of these patients find a strange remedy in opium. My attention was first directed to this means by the case of a woman who was under the care of my friend, Dr. Litteljohn. The bowels were obstinate, and Dr. Litteljohn prescribed half a grain of opium to lessen the pain of an inflamed ovary. To his astonishment after the opium she got a natural evacuation, which she had not had for a long time.

In this patient the action of the bowels clearly appeared to be inhibited by the reflex irritation of the splanchnics, or by irritation of the ovarian nerves; and in other cases I have little doubt that a similar inhibition of the intestinal movements takes place from ovarian irritation due to walking. When the patient is at rest, on the contrary, the irritation is prevented, the inhibition of the intestine ceases, and natural movement occurs. In the same way inhibition of the intestines may occur from piles, fissure, fistula, or other disease of the pelvic organs; not only may the voluntary expulsive efforts of the patient be diminished by fear of the pain which the passage of a motion may cause, but also the movements of the intestine itself may actually be lessened.

Increased resistance to evacuation may also occur from adhesions of the intestine due to old peritonitis; from constriction by bands passing over them, or by contraction of the lumen from cicatrisation of old ulcers, or the presence of malignant disease. Resistance may also occur by pressure from without, as by a retroverted uterus; by a uterine or ovarian tumour; by an enlarged prostate, or by other growth in the pelvis. In some cases a lessening of the lumen of the upper part of the rectum may occur from a tendency of the sigmoid to prolapse into it; though this condition is one which more frequently gives rise to diarrhœa.

Symptoms.—The distinctive sign of constipation is that the evacuations occur too seldom, or are too scanty. The amount of evacuation

daily passed by a healthy person depends very much on the quantity and quality of the food; but ordinarily the amount passed, on a mixed diet, forms a brown cylindrical mass about five to eight inches long and an inch to an inch and a half in diameter. In constipation the amount may not be half of this; and consists of small dry, hard lumps, sometimes quite round, and of the size of hazel-nuts or walnuts. In some persons the omission of a motion, or its absence even for several days together, seems to cause no symptoms, either local or general; but usually in a healthy person, if the desire to defæcate be checked, the impulse to evacuate the bowel may indeed soon pass off, but it is frequently succeeded by a feeling of discomfort in the abdomen, and a sense of heaviness and dulness in the head which may amount to an actual headache. If the bowels still be not evacuated these symptoms may pass off, and remain absent for a day or two; but when the bowels continue constipated the hardened fæces may act as a mechanical stimulus to the bowel and cause diarrhœa, which brings relief. Or, instead of causing a single attack of diarrhœa, the scybala may remain and give rise to catarrh of the colon, showing itself in alternate constipation and diarrhœa; or they may produce the symptoms usually associated with gastro-intestinal catarrh, namely, loss of appetite, furred tongue, disagreeable smell of the breath with colicky pains, yellowness of the conjunctiva, or, if the duodenum be much implicated, even actual jaundice. Along with these symptoms, referable to the gastro-intestinal canal, may be associated dulness of intellect, irritability of temper, and depression of spirits with a tendency to headache. Continuous and obstinate constipation has been charged with causing still more serious cerebral disturbance, such as giddiness, hypochondriasis, and even melancholia or mania, symptoms which have disappeared on a proper relief of the bowels. Palpitation may also result from constipation, but probably this affection is not due directly to the fæcal matters in the intestine, but rather to the flatulent distension to which accumulation of fæces gives rise. The local symptoms will depend on the site and size of the accumulation. If present in the cæcum or ascending colon, it will give rise to pain and tenderness, and may lead to typhlitis with consequent peritonitis (see p. 923). When it occurs in the sigmoid it will press upon the pelvic viscera, and by pressure on the bladder may bring about frequency of micturition and nocturnal enuresis; by pressing upon the uterus it may give rise to dragging pain and disturbance of the menstrual functions, dysmenorrhœa, amenorrhœa, or menorrhagia; by pressing on the seminal vesicles it may cause nocturnal emissions. The so-called seminal emissions occurring during defæcation are generally due in part to the mechanical pressure of scybala, and in part to the increased straining which is necessary to evacuate them. When fæcal accumulation is very great it may give rise to complete obstruction of the intestine and eventually to death.

Treatment.—*Habit.*—One of the most important factors in the treatment of habitual constipation is the acquisition of the habit of regularly evacuating the bowels at the same time every day. In order that this

habit should be formed the patient should go to the closet every day at the same hour; and he should do so, if possible, by the clock with perfect precision, whether he anticipate an evacuation or not.

Time.—The time chosen should be such that it is not likely to be interfered with by avocations. In most cases the best time is the morning just after breakfast; but if the patient is likely to be unable to keep to a definite hour in the morning, it is better to fix the evening just before going to bed. The evening should also be chosen for evacuation by those who suffer much from piles or tendency to prolapse; because if the bowels are open in the morning the piles are apt to become full and congested, or the prolapsed bowel to come down and give discomfort and trouble to the patient during the whole day. On the other hand, if the bowels are evacuated just before going to bed, the parts can be washed, the piles or prolapse may be returned, and, the recumbent position being then kept for many hours, little or no discomfort will be felt by the patient. Thus the natural tendency to recovery from these diseases will be aided. While at stool, it is sometimes advisable to aid the passage of the fæces from the sigmoid into the rectum by pressing the abdomen with the ball of the left thumb over the course of the bowel from the left margin of the ribs, between the umbilicus and iliac crest, down towards the pubes. If, after a few attempts, no desire be felt, one or two drachms of glycerine may be injected into the bowel, so as to give the local stimulation to its action which fæcal matter present in the rectum would usually exert. The stimulation thus afforded is frequently sufficient to start the peristaltic action of the bowel, and to bring the fæces from the sigmoid into the rectum; as well as to call into play by reflex action the abdominal muscles which usually aid an act of defæcation.

Position.—The practice adopted in this country of sitting on a closet is unfavourable to the evacuation of the bowels. If the individual sit upright the nates may be pressed together, and thus afford a mechanical impediment to the exit of the fæces. Many persons avoid this difficulty by bending the body forward at an acute angle. This position has the further advantage of putting the floor of the pelvis on the strain, so that it aids in turning the fæcal masses forwards out of the rectum through the anus. Such stretching is more especially necessary in persons whose abdominal muscles are flabby and lax; especially in women who have borne several children. The crouching position in which defæcation naturally takes place in the open air, and in most countries, may be partially imitated in a water-closet by having a high footstool; but when there is much difficulty in evacuating the bowels it is still better to let the patient sit over an ordinary chamber-pot on the floor. In cases in which the uterus is retroverted or enlarged this position has the further advantage of allowing this organ to fall forward, so that its backward pressure may not interfere with the passage of fæces through the rectum.

Diet.—Dietetic treatment is of great importance in constipation.

Oatmeal porridge for breakfast is a good laxative, and its action is still further increased if golden syrup or treacle be taken with it instead of milk. Various kinds of bread have been specially recommended for constipation, the essential point in each of them being that the flour is not so finely ground, nor the starchy particles of it so completely separated from the harder outside of the grain as in fine white flour. Amongst the various kinds used are varieties of whole-meal bread, bran biscuits, and admixtures of whole meal and bran in different proportions. Golden syrup and treacle still retain their laxative properties when mixed with flour, so that various kinds of ginger-bread have a laxative action.

All vegetables, or fruit, containing much cellulose, especially in the form of hard fibres, leave a good deal of indigestible residue in the intestine; hence they have a somewhat aperient action. Green vegetables of various kinds—cabbage, spinach, Brussels sprouts, asparagus, Portugal onions, carrots, haricots, and turnips—are all useful; and some of them, especially cabbage, tend to act as laxatives not only by the mechanical effect upon the intestine, but also by forming a good deal of gas in the intestine. This gas is occasionally so voluminous as to become objectionable and to interfere with the use of the vegetables. Besides these vegetables, which are usually eaten cooked, there are some, such as tomatoes, which are useful both cooked and raw. A tomato eaten at breakfast will sometimes keep the bowels regular; and a raw apple eaten in the morning before breakfast has a similar effect. Stewed fruits, gooseberries, apricots, apples, pears, plums, and especially prunes, have all a laxative action, and may be used at meal-times for this purpose. Baked apples and stewed prunes are most commonly preferred. They probably owe their action not only to the cellulose that they contain, but to the sugar and neutral salts which are present in them. Jams and jellies made from fruit have also a laxative action. In jams there is the mechanical effect of the seeds or skins which they contain; in jellies this is absent, but the sugar and the vegetable salts render them slightly laxative, though not so much so as jams or marmalade. Syrup and honey have also a laxative action; but one of the best substances of this kind is marmalade, and this addition to breakfast has greatly increased in popularity.

Drinks.—The amount and kind of water drunk is also of great importance. Many people drink too little water, and frequently a tumblerful of cold water on rising in the morning, or on going to bed at night, or on both occasions, suffices to keep the bowels open. In winter, and with delicate persons in whom the cold water tends to lie heavy at the stomach, a tumblerful of hot water sipped on rising in the morning and on going to bed at night is better than cold. To the constipating effects of hard water I have referred more than once, and people who live on a chalky soil do well to remove the lime from the water, as far as possible, by boiling it first, and after it has settled, or has been filtered, exposing it to the air so that it may regain its freshness; or it may be aerated in a gasogene. If the water, instead of carbonate of lime which is removed by

boiling, contain sulphate of lime which remains in solution notwithstanding the boiling, so that the water is permanently hard, patients who are troubled with constipation should drink aerated water, and make tea of rain water or again of some kind of aerated water.

Teas and wines vary so much in their character that while some of them tend to produce constipation, others rather tend to act as laxatives. As a rule, however, tea contains a considerable proportion of tannin, which may act as an astringent; so that although in some persons tea is a laxative, yet in cases of constipation cocoa or coffee should be tried instead. Some of the red wines, such as certain clarets or ports, contain much tannin and are very astringent; white wines are less so, and in constipation are therefore to be preferred.

Exercise.—As I have said, want of exercise tends to cause constipation, and active exercise to relieve it; and that the active exercise ought to be sufficiently brisk to induce active play of the diaphragm and abdominal muscles. The languid saunter along a road, which is sometimes known by the name of a "constitutional walk," is of comparatively little service in this respect. I repeat, however, that sometimes exercise, as in cases of ovarian or uterine irritability, has just the opposite effect.

Massage.—In chronic constipation massage sometimes proves a very useful means of treatment. It may be applied to a certain extent by the patient himself, who may gently rub the abdomen in the direction of the hands of a watch every morning or night, or may roll a cannon ball (from 4 to 7 lbs. weight, and covered with chamois leather) in the same direction. Seven pounds seems a great weight when held in the hand, but a cannon ball of this weight does not feel at all too heavy when it is rolled over the abdomen. These simple methods, however, are by no means so efficacious as the regular application of massage by a trained operator.

Hydropathy.—In some cases hydropathic treatment is very useful [*vide* vol. i. p. 345]. The essentials of the hydropathic treatment are the application of wet compresses to the abdomen two or three times a day and sitz baths—cold in the summer and the chill taken off in winter. These must be followed or accompanied by exercise and drinking water; so that it is almost impossible to get the full effect of the therapeutic measures in a patient who is being treated at home.

Drugs.—The drugs that have been used for the relief of chronic constipation are many indeed; but, on account of the important part which inaction of the rectum plays in the malady, aloes, which has an especial action upon that part of the bowel, is more universally employed, either alone or in combination, than any other, in the medicinal treatment of constipation. It forms a part of every purgative vegetable pill in the Pharmacopœia with the exception of the compound scammony pill. When given alone it is convenient to administer it in the form of granules which contain one-tenth of a grain each of aloin made up with sugar and milk. One of these granules taken with each meal suffices to keep the bowels regular. One objection that is frequently raised against aloes is that it tends to cause piles; and there can be no

doubt that a full dose of an aloetic purgative is very likely to bring on an attack of piles in those liable to this ailment. But small doses of aloes, on the contrary, by maintaining a regular action of the bowel, tend to prevent piles by avoiding the local pressure in the rectum which large and hard motions occasion. In combination with rhubarb or colocynth, aloes is employed as a dinner or breakfast pill, and such a formula as the following is very useful:—℞ Pil. colocynth co., Pil. rhei co., āā gr. j.; Ext. hyoscyam. gr. ss. M. Ft. pil.

An old friend of mine, to whom I owe this formula, told me that he had used it every night of his life for forty years; it had done him no harm: on the contrary, it had done him much good, and the long continuance of it did not seem to diminish its effect nor had the dose to be increased. He took his pill every day five minutes before dinner, so as to give it a slight start of the food, and this plan, as judged by its results, seemed to be a good one. Where the pill taken at night does not act quickly enough so as to produce a motion in the morning at the time that is usually most convenient to the patient, it may be taken at lunch or even at breakfast. The three remedies that are most in vogue at present as laxatives are the sulphur lozenges of the Pharmacopœia, which we owe to Sir Alfred Garrod, compound liquorice powder, and cascara sagrada. Two or three sulphur lozenges every night frequently suffice to produce a soft pultaceous action next morning; and half a teaspoonful to a teaspoonful of liquorice powder in half a wineglassful of water has a similar action in cases where the sulphur alone is insufficient to produce the effect. Cascara sagrada, in the form of the extract or liquid extract, has largely displaced other purgatives. The solid extract is the more convenient, as it may be given in pill in doses of one to three grains either alone or combined with extract of hyoscyamus, or with extract of hyoscyamus and some hepatic stimulant, such as euonymin or iridin. A plan which is useful in some cases is to put a pinch of senna leaves or pods in a small muslin bag, and place this in a stewpan with some water and prunes. During the process of stewing the active principle of the senna is dissolved by the water, and imparts stronger aperient power to the prunes and their juice. The bag is removed when the prunes are stewed, and a little experience teaches the patient the proper quantity of the confection to use. In atony of the intestine strychnine or nux vomica is a useful adjunct; and belladonna not only tends to prevent griping, but seems to have a regulating action upon the nervous system of the intestine itself. In one case of obstinate constipation I succeeded in obtaining natural action for some months by the use of a pill containing extract of hyoscyamus and extract of nux vomica without any direct purgative whatever. The idea of using such a pill in chronic constipation was to try to shift the balance of excitability from the inhibitory to the motor nerves of the intestine. In another case of obstinate constipation in a gouty patient, I succeeded in obtaining regular action of the bowels, which had previously resisted very powerful purgatives, by putting the patient on a course of salicylate of soda. In some patients meat and beef tea appear to have a strongly

constipating action; in others they have just the reverse. In many patients the omission of meat from the diet table causes the bowels to move regularly—a result not due simply to an increased bulk in vegetable diet, for in some cases it is obtained when the diet remains otherwise almost unaltered.

In some cases, again, bread seems to have a very constipating action, and one patient, who suffered from obstinate constipation, told me that since he gave up bread entirely and lived upon fish, meat, and vegetables, his bowels have been perfectly regular.

The addition of iron appears sometimes to aid the action of aperient pills; and the pill of aloes and iron of the British Pharmacopœia sometimes acts very satisfactorily. When one aperient ceases to act another may be given; and by changing the purgative from time to time regular action of the bowels may be maintained. Many people think that the continued action of purgatives is unnatural and injurious, but, at any rate, the harm they do is less than is likely to occur to the patient from accumulation of fæcal matters in the intestine; and dinner pills may be simply looked upon in many cases as supplying the stimulants of which the food has been deprived during the process of cooking. If there be any large accumulation of hardened fæces in the bowels the fæces should not be driven down forcibly by a violent purgative, but should be washed out from below by means of an enema. Enemas consisting of warm water alone, of soap and water, or of castor oil and starch mucilage, are frequently employed, not only to remove hardened fæcal masses but also to induce daily action of the bowels. It is usually said that their continued use is apt to produce a catarrhal and atonic condition of the intestine; moreover they are troublesome and often inconvenient to the patient. Of late years enemas of one or two fluid drachms of glycerine, or suppositories containing glycerine, have been employed to cause evacuation of the bowel; and although they too are liable to lose their power, they are frequently very useful indeed, because the stimulus to defæcation comes from the rectum; if no fæcal matter is present no desire is felt. The advantage of glycerine is that, if a patient on going to the closet is unable to evacuate, by using glycerine he will probably get the desire, and obtain evacuation in about ten minutes, instead of waiting until next morning to obtain evacuation by means of a pill or aperient draught.

When large masses of scybala have accumulated in the intestine, it may be necessary to remove them mechanically, by breaking them down with the handle of a spoon if they are in the rectum, or by washing them out if they are higher up. In order to do this they should be softened as far as possible by the introduction of large enemas having either oil or water as a basis. Olive oil may be used alone, a pint or more being thrown up if the patient be able to retain it. Water at about a temperature of 98° F. should be used for the enema, as either cold water or very hot water tends to increase the action of the bowel. It should be introduced under very gentle pressure indeed, either with a

syringe or, still better, by an irrigator, which should be suspended twelve or eighteen inches above the patient's hips. The exact height must be regulated by the person's feelings; if the bowel be very irritable the pressure must be diminished; if it be but slightly irritable, the pressure may be increased. As large a quantity of water as can be retained should be thus introduced—usually from one to three pints; and it is often retained better when introduced through a soft rubber tube, not directly into the rectum, but into the sigmoid flexure. The size of the tube must be regulated according to the case; but frequently a stout tube half an inch in diameter is better than a small one, as it is not so likely to kink in the rectum, and the end of it passes more easily into the sigmoid flexure. Linseed tea, or thin starch paste, may be employed instead of plain water. In order to allow of the introduction of such large quantities into the bowel, it is advisable to pass the rectum tube through a piece of dentist's rubber which may be held tightly against the anus, either by the fingers of the nurse or by a napkin folded round it. When desire to defæcate comes on during the introduction of an enema its administration should be stopped for a few minutes, and gentle pressure on the anus kept up. The desire to defæcate then usually passes away, and its cessation may be aided by very gentle rubbing over the abdomen. After the liquid has been introduced, it is well to keep a folded napkin over the anus with a certain amount of pressure for some time, so that the liquid may be retained and gradually soften the scybala. If the bowel be very irritable a dose of opium may be given—either by the mouth or, still better, as an opium injection, or as a morphia suppository—half an hour before the introduction of the softening enema.

In some cases, where fæcal concretions are very hard to remove, the action of the enema below may be aided by washing out the stomach. This operation seems to exercise a stimulating action through the nerves on the movements or secretions of the intestine, and thus aids in the removal of the fæcal mass.

T. LAUDER BRUNTON.

DISEASES OF THE SMALL INTESTINE

UNDER this heading reference will be made to those morbid conditions only which have not been described elsewhere, lest any be omitted from the *System of Medicine.* In many instances the lesions of the small intestine have been described under other headings, such as typhoid fever, intestinal obstruction, and so forth ; and a cross reference is all that is required.

SYNOPSIS

Malformations.—Duodenal pouches ; duodenal occlusion ; Meckel's diverticulum ; occlusions of small intestine.
Innocent tumours of duodenum and small intestine.
Lymphadenoma.
Vascular disturbances of intestine.
Ulceration of intestine : tuberculous, syphilitic.
Malignant growths. — Primary : (*a*) in duodenum ; (*b*) in jejunum and ileum. Secondary.

MALFORMATIONS.—**Duodenal pouches.**—The duodenum occasionally presents a small pouch arising from the second part, just above the biliary papilla ; the mouth of the pouch is wide, and the depth of the pouch about one inch. The walls are formed of the normal coats of the intestine, and though they are thin there is no reason to believe that the condition is due to ulceration. The pouches are usually supported by the pancreas, and probably this is why they remain of comparatively small size. The biliary papilla may be just within the margins of the pouch, but the pouch is not the end of the common bile-duct greatly dilated. The constancy of its position makes it probable that the pouch is a congenital imperfection connected with the adjacent hepatic diverticulum. It is noteworthy that congenital occlusion of the duodenum is situated at the same level, and it is probable that both these defects are the expression of departures during fœtal life from the normal development of the hepatic diverticulum. The departures appear to be in opposite directions, and it is interesting to note that somewhat analogous abnormalities may occur in the ileum in connection with the vitelline duct, which may persist as Meckel's diverticulum, or, not content with self-obliteration, may give rise to occlusion of the ileum. I have seen the association of a duodenal pouch and of a Meckel's diverticulum in the same body.

No symptoms or evil results are known to be due to duodenal pouches ; it is conceivable that, if filled with food, they might exert pressure on the common bile-duct. Pertik records a glove-shaped diverticulum which, as it became filled with food, must have exerted pressure on the lumen of the duodenum and narrowed it ; but, as Ewald says, this is probably a unique case.

Congenital occlusion of the duodenum occurs just above the entrance of the biliary papilla into the duodenum. There may be a diaphragm with

or without a perforation in the centre running across the bowel, or the continuity of the bowel may be completely interrupted. The septum is composed of the muscular as well as of the mucous and submucous coats of the bowel; its position is constant, and points to the conclusion that it is connected with the budding-off of the hepatic diverticulum in fœtal life. Shattock explains the mechanism both of this congenital defect and of the analogous one of congenital atresia of the œsophagus, by supposing that the unequal growth which leads to the formation of the glandular diverticulum (liver or lungs) produces a kink on the opposite side of the original tube, much as will happen if the intestine be drawn out in an artificial anus through any thickness of the abdominal wall, and that this displacement may conceivably lead to secondary closure of the original tube.

The part of the duodenum above the occlusion becomes dilated, and may form a tense thin-walled cyst; this part of the duodenum may end blindly as a cul-de-sac, or, as is generally the case, it remains connected with the third part of the duodenum.

The dilatation spreads from the first part of the duodenum to the stomach.

If the occlusion be complete, vomiting begins soon after birth and continues until death, which usually follows within five days. The complete absence of bile from the vomit should suggest this form of congenital occlusion rather than one lower down, below the entrance of the bile-duct. The vomit may be clear at first, and subsequently of coffee-ground character. Meconium passes into the fæces, and may be voided per rectum. Jaundice is absent unless some secondary disturbance be set up, as occurred in the case recorded by Champneys and Power.

When the occlusion is not complete some food may pass through into the intestine, and life is not so rapidly brought to a close.

Ducros refers to congenital narrowing, obliteration, and complete interruption of the alimentary canal at the junction of the duodenum with the jejunum. He collected three cases of stenosis, five of obliteration of the lumen, and three of complete interruption of the intestine at this point. The probable cause of such changes in this situation is pressure from the anatomical arrangement of the peritoneum at the duodeno-jejunal junction.

Meckel's diverticulum is the persistence of the omphalo-mesenteric or vitelline duct. It occurs in from 2 to 3 per cent of bodies examined, and usually comes off the ileum within three feet of the cæcum; it may arise much higher up, but the pouches occasionally seen in the duodenum should not be called Meckel's diverticula since, as we have already seen, they are connected rather with the hepatic offshoot from the intestine than with the vitelline duct. It appears to occur more commonly in males. There is considerable variation in the attachments, size, and shape of the diverticulum. It is commonly about 2 to 3 inches long, and about the same lumen as the ileum from which it springs; it may, however, be much longer. Pollard describes an exceptional case in which the intestine gave off a diverticulum

36 inches in length at a distance of 24 inches from the pylorus, the continuation of the small intestine being 63 inches long; on the other hand, it may only be represented by a slight pouching of the intestine.

It may run to the umbilicus and end blindly in the umbilical cord, in which case it will be cut through at birth, and give rise to an intestinal fistula; it may be adherent to the internal surface of the umbilicus, or be attached to that spot by a fibrous cord of varying length. When opening widely at the umbilicus the mucous membrane of the diverticulum may prolapse, sometimes so extensively that a spur from the ileum projects at the wound with two openings into the ileum, one on each side of it. Golding Bird describes a case in which the ileum below Meckel's diverticulum became intussuscepted into the diverticulum, and refers to a case of Barth's in which the proximal piece of the ileum passed into the diverticulum and formed an intussusception.

When the diverticulum opens at the umbilicus, pouching of the mucous membrane may give rise to congenital umbilical polypi microscopically resembling the mucous membrane of the diverticulum. Such polypi may be found without any direct evidence that a diverticulum is attached at or lying open at the umbilicus. It may be noted that other forms of umbilical polypi, composed of granulation or angiomatous tissue, may occur in children. Cysts lined with columnar epithelium close to the umbilicus may be derived from the vitelline duct.

To return to its attachments; it may be adherent to the inner surface of the umbilicus, or be tethered to it by fibrous cord, which represents its atrophied continuation. This tag may become loose, and may then become adherent to the peritoneum elsewhere.

The various ways in which strangulation of the intestine may be brought about by the diverticulum or the fibrous band arising from or representing it are fully described in the article on intestinal obstruction. Most frequently the diverticulum ends blindly, and its termination is usually smooth and rounded, but, as in a case figured by Hudson, it may be knobby and hammer-shaped.

The diverticulum may project from the convexity of the ileum, and lie quite free, or be provided with a mesentery of its own; or, again, it may lie between the layers of the mesentery arriving from the concavity of the intestine. Its walls are like those of the ileum, and it may share in the morbid conditions affecting the intestine; thus it may be strangulated in a hernial sac, or be attacked by various forms of ulceration, and in one remarkable example recorded by Makins there were no less than thirteen perforations in it. Inversion of the diverticulum into the lumen of the ileum has been known to give rise to intussusception; and it is possible that invagination of a small diverticulum and subsequent changes taking place in it may explain the presence of some polypi found in the lower part of the ileum (*vide* p. 713).

The part which the vitelline duct plays in the production of congenital obstruction of the ileum will be referred to later.

Multiple diverticula of the small intestine are very rare, they are not

derived, like the single Meckel's diverticulum, from the vitelline duct. They resemble the sacculi in the colon found between the longitudinal muscular bands. They seem to occur chiefly along the mesenteric attachment. They have been attributed to damage received in a hernial sac, though evidence of this may be wanting. Perhaps they may be due to a deficiency of the longitudinal muscular coat allowing a series of herniations of the intestinal walls; or possibly they may be due to fœtal peritonitis drawing out and partially separating small pieces of the intestinal wall. Complete separation of numerous bits of the intestinal wall by this process of fœtal peritonitis has been thought to explain the presence of multiple cysts in connection with the intestine; a slighter degree would give rise to sacculi.

Occlusion of the small intestine congenital in origin may be complete; the lumen of the intestine being interrupted, and the intestinal walls only represented by a cord connecting the pervious parts of the bowel. These occlusions may be multiple. They are of course incompatible with life for more than a few days.

In other instances the small intestine may be partially stenosed by contraction of its walls, or may be obstructed by a diaphragm stretched across its lumen like that seen in partial occlusion of the duodenum. This narrowing is most frequent in the lower part of the ileum, in the situation, that is, where the diverticulum ilei occurs.

Sometimes a fibrous band representing the remains of the vitelline duct is seen to be attached at the point of the stricture; in such cases the stenosis might be mechanical and due to the intestine winding the band round itself; but such cases are exceptional. More often the stenosis is unaccompanied by any remains of the vitelline duct.

The most probable explanation of the presence of single obliterations, narrowings, or diaphragmatic membranes in the ileum is that the process of atrophy and involution of the vitelline duct has become excessive, and by spreading to the intestine at the point of junction has produced similar changes there. A similar pathological process may occur in the aorta at the situation of the ductus arteriosus, or lower down where the right aortic arch is attached in fœtal life.

In other cases similar but multiple narrowings of the small intestine are found. They show no sign of past ulceration or other morbid process, and though less advanced are of the same pattern as the multiple complete obliterations mentioned above. They are presumably due to the same cause. Whatever it may be it is active in fœtal life. It does not seem probable that excess in the obliteration of the vitelline duct would explain multiple obliterations, especially when they are far apart. Such an explanation could not possibly be stretched to fit obliterations on the same body so distant as in the upper part of the jejunum and lower part of the ileum.

Intra-uterine peritonitis, by interfering early in fœtal life with the proper development of the intestine, or later by producing bands and adhesions by which the intestine is constricted and locally obliterated,

offers a possible explanation. While abnormal development of the valvulæ conniventes has been suggested as an explanation of multiple diaphragmatic strictures, the cause of this hypertrophy in the valvulæ conniventes is wanting. It is rather against this view that the valvulæ conniventes are but slightly developed in fœtal life (29).

The fact that when congenital stricture of the small intestine occurs it is comparatively often multiple, shows the multiplicity is not a mere coincidence.

Clinically, partial single narrowing of the lumen, such as may occur in the ileum near the site of Meckel's diverticulum, may give rise to no signs of obstruction, and is compatible with long life (19). I have seen a case with three diaphragmatic obstructions in a man aged twenty-five years without any intestinal troubles. In other cases there may be signs of chronic obstruction. The liquid state of the fæces in the small intestine accounts for the absence of symptoms when there is a considerable amount of narrowing of the calibre of the intestine.

Innocent tumours of the duodenum require hardly any comment. Polypi, formed from the mucous membrane and having a papillomatous structure, occur much less often here than in the rest of the intestinal tract; though exceptionally, as in Collier's case, they may be more numerous on the duodenum than in the remainder of the intestine.

Dr. Perry has described a single large papilloma arising from the mucosa around and below the biliary papilla, which did not obstruct the bile-duct.

A large myxo-fibromatous polypus arising in the second part of the duodenum gave rise, in a patient of Dr. Foxwell, to the symptoms of pyloric obstruction.

In a remarkable case of lymphangiectasis of the intestine described by Allchin and Hebb, the mucous membrane of the duodenum and jejunum were the parts most affected.

Lymphadenoma in the duodenum is so like the same condition in the small intestine that it need not be described separately.

INNOCENT TUMOURS OF THE JEJUNUM AND ILEUM. — **Polypi** of the small intestine have been sufficiently described in the article on intestinal obstruction. The papillomatous glandular or mucous polypus is the commonest.

Large polypi containing muscular or fibrous tissue may obstruct the lumen of the gut or give rise to intussusception.

The Guy's Hospital Museum (No. 891) contains a polypus of the ileum with a central mass of fat, and was thought to be due to invagination of a Meckel's diverticulum taking some of the mesenteric fat in with it.

Innumerable villosities due to lymphangiectasis have been described, and in a minor degree dilatation of the lacteals is not so very rare, and has been found in association with chylous ascites.

Multiple congenital cysts of the intestine have been explained on the hypothesis that fœtal peritonitis cuts off small portions of the intestinal

walls, and that these fœtal inclusions subsequently undergo cystic dilatation with mucoid contents. Roth has found an intestinal cyst lined with ciliated epithelium, an observation bearing in a suggestive manner on the character of the intestinal mucosa in early fœtal life.

In a man who died of phthisis, Letulle found 300-400 cysts of various sizes in the submucous layer; the larger ones contained fluid, the smaller ones leucocytes and giant cells, but as no tubercle bacilli could be found he did not regard them as tuberculous, but as derived from the mucosa.

Lymphadenoma is not very commonly seen in the intestinal tract. Dr. Newton Pitt has given an admirable summary of the pathological conditions, which he divides into—(i.) Lymphadenoma attacking the lymphoid tissue in the mucous and submucous coats, and giving rise to tumours which, though projecting into the lumen of the bowel, do not materially alter its calibre; usually they do not ulcerate. The lower part of the ileum around the ileo-cæcal valve is most frequently attacked, the duodenum rarely, and the colon often escapes.

(ii.) Lymphadenoma arising in the subserous lymphatics, and spreading from the mesenteric glands. The intestine is attacked from without, and becomes enclosed in a sheath of lymphadenomatous growth which invades the muscular coat, paralysing it, and thus leading to dilatation of the bowel. The growth is not so well supplied with blood as in the first case; and when it penetrates to the mucous membrane it is therefore more liable to ulcerate. It does not, like the first group, specially attack the Peyer's patches and solitary glands.

Roughly speaking, lymphadenoma seems more prone to attack that part of the alimentary canal, the small intestine, which primary carcinoma avoids; they might be said to divide it between them. Thus Pitt draws attention to the fact that lymphadenoma of the duodenum may be limited abruptly by the pylorus, and that it may also avoid the pylorus, a frequent site of primary carcinoma, in spite of the fact that lymphoid tissue is found there in young people.

Lymphadenoma of the intestine, especially when it spreads in from the outer surface, may give rise to severe or intractable diarrhœa, and this fact should be borne in mind; otherwise the diarrhœa might be put down to the administration of medicinal doses of arsenic.

VASCULAR DISTURBANCES in the intestines are of very various kinds. —The chronic venous engorgement of portal stagnation, whether this be due to tricuspid regurgitation or to obstruction in the liver as in cirrhosis, easily passes into catarrhal inflammation, and is then characterised by the presence of tenacious mucus on the surface of the intestine.

Localised dilatation of veins is sometimes seen in this condition, but may occur independently.

Active congestion may, of course, be seen post-mortem in toxic cases of enteritis and diarrhœa, but it is frequently absent when, from clinical evidence, it might have been expected.

Hæmorrhages into the substance of the intestinal mucous membrane or into the submucous coat occur in the infective endocarditis from embolism, sometimes in thrombosis, in some cases of chronic Bright's disease giving rise to the albuminuric enteritis described by Dr. Dickinson (*vide* article "Colitis"), in purpura, where again ulceration has been noted, in strangulation of the bowel, and in other cases where the cause may not be evident. In the last case the hæmorrhage may be extensive and suggest at once embolism or thrombosis of the mesenteric vessels, but careful search may fail to bring such a cause to light; some of these cases seem to be allied to the sanguineous peritoneal cysts described by Dr. T. Fisher.

ULCERATION OF THE INTESTINES is described under so many other headings, that it would weary the reader, and, indeed, be unnecessary, to give all the cross references to the diseases of which ulceration of the small intestine is either an integral part or a more or less direct result.

Generally speaking, reference should be made to the articles on Diarrhœa, Diarrhœa in Children, Colitis.

An account of tuberculous ulceration and some remarks on syphilis and its relation to intestinal ulceration have been appended.

Tuberculous ulceration.—*Modes of attack.*—As in other parts of the body in generalised tuberculosis, miliary tubercles may occur in the mucosa of the intestine, but the deposit is, of course, of no clinical importance.

Primary infection of the mucous membrane from tuberculous milk or meat contaminated with tuberculous material, or even from butter, occurs very rarely in adults; more commonly in children.

Secondary tuberculosis of the intestine due to the swallowing of sputum containing tubercle bacilli is very commonly seen, and has been found in over 50 per cent of fatal cases of phthisis. It must be borne in mind, however, that tuberculous patients may suffer from follicular ulceration of the small intestine, which may well be due to the pyococci contained in the sputum they swallow; and that every ulcer found in the small intestine of a patient dying from tuberculosis is not necessarily tuberculous.

The presence of simple follicular ulcers certainly renders the intestine more vulnerable to tuberculous infection, and a secondary infection may take place. Chronic intestinal catarrh, such as that seen in rickets, alcoholism, and other conditions, is probably a disposing factor to the development of intestinal tuberculosis of considerable importance.

The first part of the duodenum is, like the stomach, quite an exceptional situation for tuberculous ulceration; this immunity is thought to depend on the acid reaction of the intestinal contents.

Infection of the mucous membrane of the intestine may occur by the extension of the tuberculous process from the peritoneum, though it is remarkable how free the mucous membrane may remain from any complication even when there is advanced tuberculous peritonitis. It is known from Martin's researches (article "Tuberculosis," vol. ii.) that

tuberculous adenitis of the abdominal glands may result from feeding animals with tuberculous sputum without any gross lesion of the intestine; from this tuberculous peritonitis may result, and finally perhaps the intestine may be infected. Tuberculous peritonitis may, however, be due to infection from other sources than the intestine (*vide* p. 659).

Tuberculous infection of the intestine might be secondary to the discharge of tuberculous abscesses or glands into the lumen of the intestine. In a case, however, under the care of my colleague, Mr. Turner, in which a tuberculous abscess in kidney burst into the duodenum, I found the intestine quite free from ulceration.

Situation.—Tuberculous ulceration of the intestine is commonest on the lower part of the ileum, and though the ulcers may extend throughout the whole of the small intestine, they become less frequent as the duodenum is approached, where they are very rarely found; ulceration has been seen, however, within half an inch of the pylorus. Since it is thought that the acidity of the gastric juice tends to prevent the occurrence of tuberculous ulceration, it is probable that in such exceptional instances its secretion was deficient in hydrochloric acid.

The ulcers, like those of typhoid fever, are more common around the ileo-cæcal valve, and may extend into the appendix giving rise to symptoms of appendicitis, or into the colon. The colon may indeed be affected without the ileum being involved, while the rectum is a not infrequent situation for such tuberculous ulcers.

Anatomical characters.—The tubercles generally begin either in Peyer's patches or the solitary glands, and after undergoing caseous degeneration appear as small yellowish spots, which soften down in the centre, run into each other, and by opening into the intestine give rise to the earliest stage of a tuberculous ulcer. The lymphoid and other tissues around are swollen from extension of the inflammatory process, and the edges of the ulcer are raised. The ulcer extends by softening and discharge of the surrounding tubercles, while at the same time infiltration of the coats of the intestine is proceeding. The lymphatic vessels become affected, and local chronic peritonitis is set up. The base of the ulcer thus becomes thickened by tuberculous inflammation in its muscular and serous walls, and small white or even yellow tubercles can be seen on the peritoneal surface, which appears somewhat opaque and thickened. The base of the ulcer is thus protected against perforation, and may be considerably thicker than the healthy parts of the intestine. The danger of perforation is still further prevented by the tendency of the local tuberculous peritonitis to cause adhesions to surrounding coils of intestine. In some cases these adhesions may be drawn out into bands [*vide* article on "Intestinal Obstruction," p. 808] or may be broken across; they are then seen as filamentous tags on the outside of the intestine, and become more evident when the intestine is placed in water. The chronic inflammation may give rise to pigmentation of the peritoneal or of the intestinal surface of the ulcer; this is more often seen in the chronic oblong ulcers in the colon.

The base of the ulcer is thickened, as already mentioned, from the

presence of caseous tubercles and inflammatory tissue, and is usually roughened and irregular; sloughs are only present exceptionally.

The edges of the ulcer are raised, thickened, and not undermined.

The ulcers, which in the early stage are small and round, become larger and irregular in outline; like all chronic ulcers they tend to run transversely round the lumen of the bowel, this extension being preceded by tuberculous infection of the lymphatic vessels, the circular course of which is shown up by the presence of caseous spots. In addition, the mucous membrane may be the subject of lardaceous change [*vide* article "Lardaceous Disease," p. 263].

The histological appearances of tubercle are fully described in the article on "Tuberculosis" (vol. ii. p. 6).

Results of tuberculous ulceration.—Perforation is a rare result, and after death, it is often very difficult to be certain whether perforation of an ulcer into the peritoneal cavity has actually taken place during life or not. For with tuberculous enteritis may be combined peritonitis, and matting together of the intestines with loculi containing turbid ascitic fluid, with varying amounts of fibrin or pus-cells; and the intestines are so softened that any attempt to ascertain whether there be a perforation of an ulcer into the cavity of the peritoneum readily leads to rupture. In some cases it may be that perforation into the intestine from without has taken place either from a localised collection of pus or from a softened caseous lymphatic gland, and not in the reverse direction.

The inflamed walls of the intestinal ulcers under the conditions such as are present when there is advanced tuberculous peritonitis, may allow of the passage of micro-organisms, such as bacillus coli, without any solution of continuity, and thus a more acute peritonitis, due to secondary infection, is set up. Though this is not a gross perforation of the intestine it comes eventually to much the same thing; it leads, that is, to increased severity of the peritoneal inflammation.

Perforation does occur, but it is rare, and contrasts with what holds in acute intestinal ulceration, such as in typhoid fever.

Stricture of the small intestine, from healing of the ulcers which run round the lumen of the intestine, probably occurs more frequently than is generally thought. But the comparatively slight narrowing that results and the fluid state of the intestinal contents combine to render signs of obstruction from tuberculous strictures, apart from the effect of tuberculous peritonitis, extremely rare. The strictures may be multiple.

Symptoms.—Very frequently no symptoms are present during life, even when as seen after death there is very considerable tuberculous ulceration in the small intestine; though from their occurrence in more than 50 per cent of the fatal cases of phthisis their existence may be suspected whenever, in the course of the disease, there is diarrhœa lasting for any time, abdominal tenderness or pain. The diarrhœa may, though rarely, be accompanied by blood. It must be remembered that simple

diarrhœa in pulmonary tuberculosis may be due to lardaceous disease, and abdominal tenderness to tuberculous peritonitis.

The presence or absence of diarrhœa seems to depend chiefly on the colon being affected, since it may be absent when the ileum is extensively affected. Generally speaking, however, diarrhœa in the course of phthisis indicates ulceration of tuberculous nature.

Diagnosis.—An important factor, as already mentioned, is the existence of tuberculous disease of the lungs or peritoneum. The passage of blood, when it occurs, would distinguish it from lardaceous disease, which is very rarely accompanied by simple ulceration of the intestine. The presence of tubercle bacilli in the fæces would of course settle the question, but little value could be attached to a negative result of an examination.

In children, in whom primary tuberculous disease of the intestine may occur without evidence of pulmonary disease, the diagnosis between gastro-intestinal catarrh and tuberculous ulceration is less easy than in adults. The presence of fever, abdominal distension, enlarged glands, and wasting and debility out of proportion to the diarrhœa, together with the absence of worms and of any other cause for the condition, are in favour of tuberculous ulceration.

Treatment.—In adults tuberculous ulceration is nearly always secondary to pulmonary tuberculosis, and the lines of treatment will be found in the special article on that disease.

It is important to guard against setting up intestinal catarrh, and so bringing on diarrhœa which may prove very rebellious to treatment. Strong purgatives may have this effect, so that care is required in combating constipation in phthisis. Food of a bland, nutritious character, which can easily be digested and absorbed, is important; if need be, it may be predigested. Dr. Colman has drawn attention to the difficulty of digesting milk, as evidenced by the large amount of curd found low down in the tuberculous intestines of children, and he advocates meat or concentrated food rather than milk.

The healing of the ulcers will be impeded by intestinal fermentation or by any irritating form of food, and it is probable that creasote and the allied drugs, which are so largely given at the present time in phthisis, exert both a preventive and curative influence on intestinal ulceration. Besides the observance of careful feeding, it is important to avoid any such exciting factors as exposure to wet or cold. When diarrhœa occurs it should be controlled by opium, bismuth, and astringents, and if necessary by morphia suppositories, or enemas containing opium.

Syphilitic ulceration.—Manifestations of the syphilitic poison, though comparatively common at the two ends of the alimentary canal, is very rarely recognised in the intervening portion. Mucous patches, pharyngeal lesions, condylomata, and syphilitic stricture of the rectum are well known and frequently met with, and are described elsewhere in this *System*. But the occurrence of syphilitic ulcers in the intestine is rare, and if they have any special characters they are not sufficiently well established.

Galliard has collected a number of examples of lesions thought to be syphilitic in infantile syphilis and in the tertiary stage of the acquired disease.

In infantile syphilis gummata beginning in the submucous coat, and when large involving the muscular coat, have been described. Thickened plaques, often involving Peyer's patches, and breaking down and ulcerating, as well as minute gummata in the mucosa, have been found. The presence of undoubted syphilitic lesions elsewhere, such as gummata in other organs, the general histological resemblance of the intestinal lesions to those characterising syphilis, and the absence of any evidence that the lesions are tuberculous, point to their being syphilitic. But they are, it must be admitted, of rare occurrence. Peritonitis may result from their presence, and, very rarely, perforation of the intestine. These lesions begin in the seventh month of intra-uterine life, and may be well developed in the eighth.

In addition to the general symptoms of infantile syphilis diarrhœa and melæna may be present, and be referred to the intestinal lesions.

The treatment is that of the general disease by mercury.

Syphilitic lesions in acquired syphilis.—The affections of the pharynx and anus seem hardly to extend into the neighbouring part of the intestinal tract; and though individuals with secondary syphilis may present intestinal ulceration, there is no convincing evidence that it is syphilitic in origin.

In tertiary syphilis the intestine may become lardaceous, and in exceptional cases ulceration has supervened. Apart from the rectum and adjacent part of the colon, the question again arises whether in any case of ulceration we are dealing with syphilitic ulceration, or merely with ulceration in a syphilitic subject; in some of the recorded cases the latter alternative appears more than probable. In this connection the not infrequent incidence of tubercle in syphilitic subjects should be borne in mind.

Breaking-down gummata in the small intestine, giving rise to ulceration, have, however, been met with. It is perhaps surprising that they are so extremely rare.

Diarrhœa is a symptom of lardaceous disease, and when relieved by iodide of potassium has been attributed to specific ulceration.

Actinomycosis of the intestine usually affects the appendix (*vide* "Perityphlitis," p. 917); perhaps in some cases it begins in the ileum close to the appendix, and gives rise to much the same signs as in the appendicular form.

For intestinal worms see article in vol. ii.; for "Sprue," p. 774; for "Colitis," p. 937; for "Enteric Fever," vol. i. p. 838.

PRIMARY MALIGNANT DISEASE OF THE SMALL INTESTINE is rare, and contrasts with the common occurrence of carcinoma in the large intestine. Haussmann collected 280 cases of cancer of the large and small intestine, and of these twenty-eight were in the small intestine, so that the

proportion is often said to be ten to one, but malignant disease of the small intestine is probably very much rarer than this ratio would suggest, most of the cases of this disease being put on record from the interest attaching to a rarity, while this is far from the case with carcinoma of the colon.

Malignant disease, when it does occur in the small intestine, avoids the jejunum and attacks the duodenum or the ileum. In some statistics the ileum is credited with an overwhelming majority of the cases, but this is probably due to cases of carcinoma of the ileo-cæcal junction and valves being put down as carcinoma of the ileum; this, for example, is true of the cases described by Du Castel as carcinoma ilei. Indeed, there appear to be comparatively few cases of malignant disease of the ileum apart from the cæcal valve. The larger number of the recorded cases of malignant disease of the small intestine are found to start in the duodenum, and, according to some statistics, three-quarters of the cases of malignant disease of the small intestine occur in the duodenum.

Primary malignant disease of the small intestine may thus be conveniently considered in two categories: (i.) as it affects the duodenum; and (ii.) in the rest of the small bowel.

When in the duodenum it resembles, as might naturally be expected, the commoner disease carcinoma of the pylorus, while the symptoms of growth in the ileum or jejunum approach those of carcinoma of the colon.

(i.) **Malignant disease of the duodenum.**—*Morbid anatomy.*—It is generally held that carcinoma of the pylorus does not extend into the duodenum, and certainly the cases are very few in which it spread widely into it; but it must be borne in mind that when carcinoma attacks the pyloric region, the actual situation of the pyloric valve can no longer be defined accurately, and there is a tendency to regard the tumour and the pylorus as coextensive.

Primary malignant disease of the duodenum must be distinguished from primary malignant disease of the head of the pancreas, and from carcinoma starting in the cavity of the biliary papilla and the conduit which is common to the ductus communis choledochus and the pancreatic duct. The pathological and clinical results of malignant disease in those two situations differ somewhat from those of malignant disease of the duodenum.

Malignant disease of the head of the pancreas is practically always a spheroidal-celled carcinoma with abundant fibrous tissue. Dr. Norman Moore has described an adeno-sarcoma, and Bard and Pic a columnar-celled carcinoma, derived from the excretory epithelium of the pancreas. The former must be extremely rare, inasmuch as no other author has described it; and the latter form of growth, according to Pic, is the same as carcinoma of the so-called ampulla or diverticulum of Vater, to which reference will now be made.

Normally the common bile-duct and Wirsung's duct of the pancreas join, and the canal thus formed runs to open on the biliary papilla, and

has been called the ampulla or diverticulum Vateri. The opening of the biliary papilla is smaller than the lumen of the ampulla, but the ampulla is little, if at all, larger than the lower end of the bile-duct. Thus it does not deserve the name ampulla except when, as the result of impaction of a gall-stone or of the growth of a papilloma or carcinoma inside it, pathological dilatation takes place.

Hanot and his pupil Vincent have described carcinoma starting in this situation as distinct from carcinoma of the lower end of the bile-duct. It has been thought that carcinoma may start from the termination of the common bile-duct where it enters the ampulla, from the termination of Wirsung's duct where it enters the ampulla, or from the mucous membrane lining the ampulla. However this may be, practically it would be difficult to distinguish them one from another, inasmuch as they are all columnar-celled growths, all arise close to each other, and all obstruct the flow of bile, and probably of pancreatic juice into the duodenum. They all arise outside the duodenum, so that if they project into the duodenum they are covered by the mucous membrane of the biliary papilla. They do not arise from the mucous membrane of the duodenum, and thus differ from carcinoma starting in the duodenum near the biliary papilla—juxta-ampullary or peri-ampullary carcinoma, as it has been called.

Malignant disease of the head of the pancreas and carcinoma of the ampulla of Vater both lead, in nearly all cases, to obstruction of the common bile-duct, dilatation of the gall-bladder, obstinate jaundice, and cholæmia, while malignant disease of the duodenum generally does not give rise to icterus.

Situation of the growth.—The growth in the duodenum may be limited to any one of the three parts, or may be more extensive and invade all three.

From an examination of the cases it appears that carcinoma, which may be columnar-celled as in the colon, or spheroidal-celled, has a tendency to produce an annular stricture; while sarcoma is more diffuse, and occurs in nodules or plaques which, by eating into the muscular walls, may even lead to dilatation of the duodenum. This condition, which is doubted by Pic, is shown in two specimens in the museum of Guy's Hospital (Nos. 750, 751), and also occurs when lymphadenoma invades the duodenum from without. When carcinoma attacks the first part of the duodenum it closely resembles pyloric obstruction in its effects, while with a malignant stricture in the third part of the duodenum bile is regurgitated into the stomach. In neither of these situations need there be jaundice, unless indeed a secondary growth happens to compress the bile-duct. In a case recorded by Dr. Pye Smith, however, there were two carcinomatous growths,—one was just beyond the pylorus, the other was three inches lower down in the duodenum; the latter was regarded as having been formed by direct transference or grafting from the growth in the first part of the duodenum. As the biliary papilla opens three or four inches from the pylorus (Quain), it is probable that in this instance it was between these two growths, and that this explained the jaundice.

The carcinomatous growths may be columnar or spheroidal-celled, and are probably derived from the lining mucous membrane. Bard, however, suggests Brunner's glands as a starting-point.

Sarcoma in most of the recorded cases appears to be of a round-celled type. The name of lympho-sarcoma and the rather vague name of lymphoma have been applied to some of the published examples, but these approach if they are not examples of lymphadenoma. The loose way in which these terms are used makes it difficult to be certain how often sarcoma of the duodenum occurs. At any rate, sarcoma is rarer than carcinoma.

In twenty-one cases of carcinoma of the duodenum which I collected from various sources, especially Pic and Whittier's records, the disease was found very unevenly distributed between the sexes; there being eighteen males to three females. The average age for the disease was fifty-two years, while in the cases of sarcoma the average age was twenty-eight years.

Carcinoma of the duodenum occurs at about the same average age as carcinoma of the rectum. In 100 unselected cases of carcinoma of the large intestine the average age was 49 years; of these 100 cases 45 were rectal cancer, with an average age of 52·3 years.

The *symptoms* of malignant disease of the duodenum vary somewhat, according to the part of the duodenum attacked, and they depend also upon the degree in which duodenal stenosis is one of the consequences.

For convenience the general and common symptoms will be given first, and then the points of difference between disease on the first, second, and third parts respectively, will be referred to.

In all cases there is wasting, loss of strength, colour and appetite, going on to cachexia; hæmatemesis usually scanty in amount, and resembling that of gastric carcinoma; vomiting at variable intervals after food; absence of free hydrochloric acid as a rule, though there may be some in combination; dilatation of the stomach with peristaltic contractions, becoming more marked as time goes on, and pain in the epigastrium and right hypochondrium. The vomiting gives rise to thirst, scanty urine, and constipation; diarrhœa is rare, but when present may alternate with constipation. In a few instances melæna may occur.

Fever when present is due to auto-intoxication, the result of absorption from the ulcerated surface of the growth; but it may not come on till the last stage, and is generally absent.

The distaste for meat is said not to be so marked as in the dyspepsia accompanying gastric carcinoma.

Flatulent eructations may be troublesome, and in one case were particularly foul, smelling of rotten eggs.

Jaundice is usually absent.

When the growth is in the first part of the duodenum, or in the upper portion of the second part, but at any rate well above the biliary papilla, the symptoms are practically indistinguishable from those of

pyloric obstruction due to carcinoma; the obstruction being only a little lower down.

There is a movable tumour as in pyloric carcinoma, and dilatation of the duodenum, pyloric incompetence, and dilatation of the stomach are present. The vomited matters do not contain bile. Hayem lays stress on the observation that when the stomach is emptied in the morning, before any breakfast has been taken, the dislodged fluid is abundant and contains large particles of food which can easily be recognised by the unaided eye; whereas, when the carcinomatous growth is below the biliary papilla, the fluid obtained under similar circumstances is less copious, and holds particles of food in such fine suspension that microscopic examination may be required for their detection. He also insists on the almost constant presence of bile in the fluid in the latter case.

Hayem considers that the permanent or continuous type of Reichmann's disease, or gastrosuccorrhœa, is really due to some obstructive cause, of which cancer may be one, either at the pylorus or in its neighbourhood; and that it is not a hyperchlorhydria setting up spasmodic contraction of the pylorus.

When the growth is below the biliary papilla, or infra-papillary, the bile and pancreatic juice tend to regurgitate through the dilated duodenum into the stomach; and their presence in the vomit should arouse suspicions that the case is not one of pyloric obstruction or of cancer in the first part of the duodenum. In any case where bile is constantly present in the vomit, the ferments of the pancreas, especially the fat-transforming steapsin, may be searched for as a confirmatory sign of regurgitation through the pylorus.

The presence of alkaline bile and pancreatic juice in the stomach neutralises the hydrochloric acid of the gastric juice, which is therefore usually, though not always, found combined and not free.

The reflux of the bile into the stomach accounts for its diminution or absence in the fæces.

If a tumour is felt it is fixed, and thus resembles that of pancreatic cancer, and differs from the movable tumour of carcinoma of the pylorus or of the first part of the duodenum.

The duodenum is more extensively dilated than in supra-papillary carcinoma, and the accumulation of food in both cases may lead to the regurgitation filling up a stomach which has been emptied over-night.

When the growth occurs near the biliary papilla—juxta- or peri-ampullary—the symptoms are a varying mixture of those in the two preceding conditions. If the growth is rather above the biliary papilla, the symptoms resemble those of pyloric obstruction; while if it tends to narrow the duodenum below the entrance of the bile-duct the symptoms of course are those of infra-papillary carcinoma. The growth may extend to the biliary papilla, and by obstructing the flow of bile lead to jaundice and thus imitate carcinoma of the pancreas.

In cases where the duodenum is not narrowed, the backward pressure

and dilatation of the duodenum and stomach need not be present; though vomiting may be severe, and the aspect of the case will perhaps be rather that of gastric ulcer.

The course of the disease thus has a very general resemblance to carcinoma of the stomach. Death usually results from asthenia and practical starvation; but in a few cases has been from loss of blood due to ulceration of the pancreatico-duodenal arteries by the growth. According to Pic, who has given an elaborate account of malignant disease of the duodenum, the duration of the disease is from three months to a year.

Diagnosis.—A passing reference has already been made to the diagnosis of carcinoma of the duodenum from malignant disease in the stomach, pancreas, and adjacent parts, and to the points which differentiate malignant disease in the first part from a similar condition in the third part of the duodenum. The diagnosis of peri-ampullary carcinoma from supra-ampullary carcinoma on the one hand, and infra-ampullary on the other, is an interesting exercise, but not one of practical importance, as there are many possible disturbing factors. Thus if the growth be in the neighbourhood of the biliary papilla, jaundice would be likely to supervene; but this is far from being constantly the case, even when the papilla itself is invaded. In a case where icterus was produced in this way the condition would probably not be accurately made out before death; for jaundice together with a tumour in the situation of the duodenum, and signs of gastric dilatation, such as vomiting, would closely resemble the commoner association of pyloric carcinoma, with a secondary growth involving the bile-duct, and so would naturally be diagnosed rather than peri-ampullary carcinoma of the duodenum.

Malignant disease of the first part is practically indistinguishable from pyloric carcinoma; and, like it, has to be diagnosed from gastric ulcer, simple dilatation of the stomach, cicatricial contraction of an ulcer near the pylorus, extensive compression of the pylorus or first part of the duodenum by tumours or by inflammatory adhesions due to cholecystitis, and possibly from hepatic cirrhosis.

Duodenal carcinoma, it is true, runs a more rapid course than many cases of scirrhus of the pylorus, but this factor would not be of much use in diagnosis.

The absence of free hydrochloric acid, which generally occurs in gastric carcinoma, is frequently found also in duodenal growths, and does not help in the differential diagnosis.

From gastric ulcer the absence of localised tenderness, the scanty hæmatemesis, the relation of pain to the ingestion of food, and the presence of a tumour would distinguish it. The presence of a tumour and the cachexia would separate it from simple dilatation of the stomach; while these points, together with absence of history and of other concomitant facts, would prevent confusion with cirrhosis of the liver. The history of cholelithiasis and the slow progress of the case should point to peritoneal adhesions around the pylorus; while the special characters of a tumour, such as an abdominal aneurysm, a cancerous or calculous gall-

bladder, or a hydronephrosis, would distinguish them from the tumour of carcinoma of the pylorus or first part of the duodenum.

The distinctions in the vomited matters of pyloric obstruction and of carcinoma of the third part of the duodenum respectively have been referred to; especial stress must be laid on the presence of bile in the vomit as disproving pyloric disease.

When obstruction occurs lower down in the small intestine or in the colon, and gives rise to obstruction, the abdomen will become more generally distended than in duodenal stenosis, where the stomach is dilated but not the small intestines.

The diagnosis from cancer of the head of the pancreas and from carcinoma attacking the lower end of the bile-ducts is easy, as in those conditions there is no obstruction in the duodenum, no dilatation of the stomach, and but little vomiting; while jaundice forms the prominent note of the disease.

Treatment is the same as that of gastric carcinoma, the medical treatment by appropriate diet by the mouth and nutrient enemas, and, if necessary, the relief of pain by hypodermic injections of morphia.

Surgically gastro-jejunostomy is of course a palliative only, but should be adopted if the vomiting be urgent, as so often it is, and if the patient be in a fairly fit state to bear the shock of the operation.

The prognosis is necessarily fatal.

(ii.) **Primary malignant disease of the jejunum and ileum.**—Primary malignant disease of the jejunum and ileum is infrequent; when it does occur it is rarer in the jejunum.

It is noteworthy that the growth may be multiple, and these are to be regarded not as independent primary neoplasms, but as examples of successful implantation or grafting of particles of malignant growth; there being always one primary growth and one or possibly more implantation growths.

Nature of growths. — Primary malignant disease of the jejunum and ileum, like that of the duodenum, may be carcinomatous or sarcomatous. Carcinoma is the commoner, and may be columnar, as in the colon; but several cases of spheroidal-celled growth are on record, and this form seems to be relatively commoner than in the colon (p. 722).

Carcinoma leads, as elsewhere in the intestine, to an annular stricture, and gives rise to chronic obstruction (*vide* article on "Intestinal Obstruction," p. 827).

Sarcoma of the small intestine is rarer than carcinoma, about twenty cases being described; many of these have been in quite young subjects.

There is a close resemblance between sarcoma of the small intestine and those examples of lymphadenoma which invade the intestinal wall from without; the chief difference is in the sarcomatous growth remaining localised to a part, or, if there be more than one growth, to parts of the alimentary canal, and not forming a diffuse or more continuous sheath as in this form of lymphadenoma.

Sarcoma of the small intestine is usually round-celled, invades the muscular coat like the variety of lymphadenoma in question, and usually leads to dilatation of the intestine at the site of the growth; sometimes, however, to narrowing. Sarcoma and lymphadenoma, then, in their local effects on the calibre of the part of the alimentary canal attacked, usually resemble acute inflammation in the softening and palsy of the muscular walls; while carcinoma, like chronic inflammation, leads to thickening, contraction, and stenosis.

Babes and Nanu have recently described a myo-sarcoma of the small intestine which was successfully removed; the man, who was thirty years of age, being in good health one year later. This case is important, since rapid metastasis is often said to occur in sarcoma of the small intestine, and to render operative interference futile.

The *symptoms* of sarcomatous growth in the small intestine are generally not, like carcinoma of the part, those of chronic obstruction, but are somewhat vague in character; pain, diarrhœa, and loss of flesh, associated with the presence of a movable tumour in the abdomen which, as time advances, tends to gravitate towards the lower part of the abdomen.

Some pedunculated tumours arising in the intestine may have the structure of spindle-celled sarcoma. They will give rise to intestinal obstruction just like carcinoma.

The only satisfactory *treatment* is excision of the growth by the surgeon; medical treatment can be but palliative.

SECONDARY GROWTHS OR INVASION OF SMALL INTESTINE.—While primary malignant disease is rare, secondary growths in and secondary invasion of the small intestine are fairly common.

When there is infection of the peritoneum with malignant disease, the intestines may become invaded from without by secondary growths. In generalised sarcomatosis the mucous membrane may contain numerous small secondary growths which may become pedunculated; in melanotic sarcoma I have seen tiny growths in the villi of the small intestine. Secondary growths, by invading the muscular coat, may draw on the circumference of the bowel and lead to narrowing, or may perhaps dispose to invagination; but as a rule secondary growths give rise to no symptoms.

The interesting process of implantation or graft growths from a growth in one part of the intestine to another has been already referred to; it forms another and probably very rare method of metastasis (26).

The small intestine may be invaded by continuity of malignant disease originating in other abnormal organs, and may become compressed and their lumen obstructed. When the invading growth starts in the colon or stomach a fistulous communication may be set up. The small intestine may become surrounded and involved in sarcomatous or other growths springing from the retroperitoneal space; thus

in one case a soft, round-cell sarcoma, encircling the first part of the duodenum, closely resembled in its effects carcinoma of the pylorus.

H. D. ROLLESTON.

REFERENCES

1. ALLCHIN and HEBB. *Trans. Path. Soc.* vol. xlvi. p. 221.—2. BABES and NANU. *Berlin. klin. Woch.* No. 7, 1897.—3. BARD and PIC. *Rev. de méd.* 1888, p. 388.—4. BARTH. *Deutsche Zeitschrift für Chirurg.* 1887.—5. CHAMPNEYS and POWER. *British Medical Journal*, 1897, vol. i. p. 718.—6. COLLIER. *Trans. Path. Soc.* xlvii. p. 46.—7. COLMAN, W. S. *British Medical Journal*, 1893, vol. ii. p. 740.—8. DICKINSON, W. H. *Trans. Royal Medical and Chirurgical Society*, vol. lxxvii. p. 111.—9. DU CASTEL. *Archiv. gén. de méd.* 1882, p. 19.—10. DUCROS. *Thèse de Paris*, 1895.—11. EWALD. *Lectures on Diseases of Stomach*, New Sydenham Society, vol. ii. p. 331.—12. FISHER, T. *Guy's Hospital Reports*, vol. xlix. p. 275.—13. FOXWELL. *Lancet*, 1889, vol i. 123).—14. GALLIARD. *La Presse médicale*, Jan. 23, 1897.—15. GOLDING BIRD. *Clin. Soc. Trans.* vol. xxix. p. 34.—16. HANOT. *Archiv. gén. de méd.* 1896, p. 547.—17. HAUSSMANN. *Thèse de Paris*, 1882.—18. HAYEM. *Medical Week*, 1897, p. 237.—19. HUDSON. *Path. Soc. Trans.* vol. xl. p. 98.—20. LETULLE. *Bull. de la Soc. Anat. Paris*, June, July 1896.—21. LUBARSCH. *Virchow's Archiv*, Bd. cxi. S. 280.—22. MAKINS. *Path. Soc. Trans.* vol. xliv. p. 90.—23. PERRY. *Trans. Path. Soc.* vol. xliv. p. 84.—24. PIC. *Rev. de méd.* 1894, p. 1081, and 1895, p. 56.—24*a*. PITT, G. N. *Trans. Path. Soc.* vol. xl. p. 80.—25. POLLARD. *Path. Soc. Trans.* vol. xlvii. p. 47.—26. PYE-SMITH. *Trans. Path. Soc.* vol. xlv. p. 63.—27. RANSOM, W. B. *Lancet*, 1890, vol. ii. p. 1021.—28. SHATTOCK. *Trans. Path. Soc.* vol. xli. p. 93.—29. SUTTON, J. B. *Trans. Royal Medical and Chirurgical Soc.* vol. lxvii. p. 157.—30. WHITTIER. *Boston Medical and Surgical Journal*, vol. cxxi. p. 377.—31. VINCENT-GEORGES. *Thèse de Paris*, 1896.

H. D. R.

COLIC

COLIC—in modern books sometimes called enteralgia—is a very old word, which originally meant a severe, sudden abdominal pain that was supposed to be due to some morbid condition of the colon. Now, for some foreign authors, it includes any sudden, sharp abdominal pain, and, therefore, it is obvious that there may be as many varieties of colic as there are abdominal or pelvic organs, or even more. The laity have given the word a still wider significance, and we find a sudden sharp pain in any part of the body called colic, for instance, a sudden pain in the head is called "head colic." Some writers have attempted to limit the use of the word according to the supposed mode of production of the pain. Thus Traube suggests that colic should mean the pain produced by the contraction of the muscular walls of any viscus, the natural issue of which is obstructed; while other authorities would limit the word to abdominal pains which, because they have no obvious cause, are thought to be neuralgic. I believe, however, that among medical men in England the word usually means a sharp, sudden pain, having its seat either in the

pelvis of the kidney, ureter, gall-bladder, biliary ducts, or intestine; hence we speak of renal, biliary, or intestinal colic; but we must remember that to say a patient is suffering from colic is not a diagnosis. For a diagnosis the cause of the pain must be discovered. Renal and biliary colic are described in other chapters, and, therefore, intestinal colic only will be considered here [see also art. "Enteralgia," p. 481].

The pain usually comes on suddenly, without warning, but occasionally it is preceded by nausea, eructations, slight intra-abdominal uneasiness or borborygmi. It is nearly always referred either to the neighbourhood of the umbilicus, or rather above it over the colon; and it may radiate in various directions about the abdomen, or even beyond it. Its most important characteristic is the griping, and the patient commonly describes it as stabbing, cutting, tearing, or boring; it is often excruciating, and the sufferer tosses about, seeking relief first in one position and then in another, but in the end usually choosing one in which he can press upon the anterior abdominal wall. Thus he may lie on his back and rub the abdomen with his hands; or he may lie on his face, pressing the abdomen against a pillow. This relief of the pain of intestinal colic by pressure is of much importance in distinguishing it from that of peritonitis, which is increased by pressure; but it must be remembered that if the colic be associated with organic disease, or great distension of the intestine, pressure may increase the pain.

Intestinal colic is paroxysmal. The paroxysm may last a few minutes, many hours, or even a couple of days; but it usually ceases as suddenly as it began, and the end may be coincident with the escape of gas from the anus. Often an attack consists of a series of frequent paroxysms; their number, duration, severity, and the length of the interval between them, are all most variable.

In a mild case of colic the pain alone attracts attention, but if it is severe other symptoms may be observed. The mere intensity of the pain may cause the patient to shriek, and even to break out into a cold clammy sweat, and then, after it has passed away, there is a feeling of weakness and faintness. The countenance is often anxious, but it rarely assumes an extreme degree of the abdominal character. Although the temperature is usually rather subnormal, it must be remembered that occasionally it is raised a degree or so, for if this be forgotten serious mistakes may be made. When the temperature is raised the pulse is proportionately quickened; but, otherwise, if the pulse be altered, it is commonly a little slowed, and its pressure and volume diminished. The abdomen is nearly always somewhat distended; and it may be possible to note the coils of intestine stretched by the gas within them, to see the peristaltic wave pass along them coincidently with the paroxysms of pain, and to observe the collapse of the intestine when, the obstruction having been overcome, the gas escapes and the pain ceases.

Very often the abdominal muscles are reflexly contracted and the abdomen becomes hard and rigid; occasionally the testicles are retracted, and Eichhorst adds that even priapism may be observed.

Nausea and the vomiting of small quantities of sour fluid are by no means uncommon; and there may be a constant desire to micturate, even with an empty bladder.

It is highly probable that the pain is due to the forcible contraction of the unstriped muscle of the intestine against an obstacle. The reasons for this belief are, that the contractions may sometimes be seen through the abdominal wall, and that the pain closely resembles that produced by the contraction of the muscle in the wall of the cystic duct, or of the ureter, when there is an obstruction due to a gall-stone or a renal calculus. It will be noticed that in both these instances the characteristic sharp, griping pain is produced by the attempt to overcome an obstacle of recent formation which may be dislodged, and that we do not usually meet with this pain when neither of these conditions is fulfilled, as, for instance, in jaundice due to the pressure on the common duct by cancerous glands in the transverse fissure of the liver. So also in the intestine, it is probable that in most cases of colic the cause of the painful muscular contraction is some obstacle which has not long been there, and which there is some chance of dislodging. This view harmonises well with the fact that ordinary colic is, of itself, rarely if ever fatal.

The commonest variety of colic is that of young children, in whom the pain is recognised by their cries, by their restlessness, and the drawing up of their legs. It is due to improper feeding, and is, therefore, much commoner in hand-fed than in breast-fed babies; it may be met with in the latter, however, when they are allowed to swallow too much milk at a time. The result of the improper feeding is that gas—due perhaps to decomposition—accumulates in the intestine and distends it, but cannot be dissipated because the presence of fæces or undigested curds prevents its passage onwards. The child is often relieved by an evacuation of the bowels, or by eructations of flatus from the mouth.

The preventive treatment consists in allowing only small quantities of food at a time; and if the child be hand-fed, lime-water or fresh barley-water should be added to each ration of milk. If even then it suffer from colic the milk should be predigested, or artificial human milk should be employed [*vide* art. "Diet of Children," vol. i. p. 412].

For immediate treatment during an attack of infantile colic warm fomentations should be applied to the abdomen; in a severe case an enema of six or eight ounces of warm water should be administered, and a grain of mercury and chalk night and morning by the mouth will be found useful. In milder cases a warmed flannel binder round the abdomen, and a mixture of one grain of bicarbonate of sodium with two or three minims of tincture of gentian in dill water, or peppermint water, sweetened with a little syrup, given three or four times a day, will generally afford relief. New-born children may suffer from colic due to retention of meconium.

In adults the most important, if not the commonest form of colic is that due to lead. This metal causes constipation, and the muscular contractions which ensue in the attempts to overcome this are particularly

prone to give rise to severe colic [*vide* art. "Lead Poisoning," vol. ii. p. 970]. It is stated, especially by German authors, that copper will produce a form of colic very closely resembling that due to lead, but it is, I believe, very rare in this country.

The simplest variety of colic seen in adults is that arising from constipation; and it is obvious that this form is analogous to the biliary or renal colic produced by gallstones or renal calculi. Its treatment is discussed under the headings of constipation and fæcal impaction.

The most characteristic colic, however, is that which is associated with certain varieties of indigestible and irritating food. Unripe hard fruits, such as apples and pears, or nuts or shell-fish in large quantities, are very common causes of it; but individual digestions differ so widely that no definite list of dangerous articles of food can be given. In these cases the colic is usually associated with constipation, and the cause of the pain is the same as in the former varieties; sometimes the irritant matter causes diarrhœa, yet even then, although the indigestible food be sufficiently irritating to set up some enteritis and consequent diarrhœa, the mass may remain, blocking up the calibre of the intestine and causing colic. The treatment is obvious; namely, rest in bed, hot fomentations to the abdomen, and a simple purge, such as castor oil, to get rid of the irritant; when the pain is severe ten or fifteen drops of laudanum may be added to it, and, should the bowels not act in a few hours, the medicine ought to be repeated. If, owing to nausea or vomiting, or for any other reason, it is inadvisable to give a purge by the mouth, a pint of warm soap and water, with some castor oil added to it, may be thrown into the rectum.

If no treatment were adopted the contractions of the intestine, after causing much pain, would nearly always overcome the difficulty in the end; the irritant would be passed and the patient would recover. The cases in which this result would not follow are those of impaction of fæces, or of foreign bodies, such as gall-stones, in the intestine; in these cases the patient is said to suffer rather from intestinal obstruction than from mere colic.

There is perhaps no ailment in which a diagnosis is more important than in colic. Many a patient who has died of peritonitis, acute intestinal obstruction or appendicitis, might have been saved had he not been treated at first under the impression that he was suffering from colic due to irritating food; and although, as Fagge says, severe colic forms an exception to Dr. Wilks's aphorism that if the pain in the abdomen is so severe as to cause the patient to send for a medical man, this in itself proves the administration of a purgative to be unjustifiable, yet the very fact that colic is the only exception should make us very cautious in our diagnosis.

Acute intestinal obstruction presents the greatest difficulty, and if the sufferer be seen early a diagnosis is often quite impossible. To such a patient a purge is nearly always administered, especially if there be any evidence that an unusual article of food has been eaten; but if this fail to

benefit him, and particularly if, as testified by frequent vomiting, by his appearance, and by the state of his pulse, he obviously and quickly becomes very ill, the purgative should on no account be repeated, for the case is probably not one of simple colic; and if it is not, purgatives are the worst possible drugs to use.

Perforation of a hollow viscus, usually the stomach, is also at first sight often regarded as colic; but it quickly either resembles intestinal obstruction or leads to obvious acute peritonitis. This accident is distinguished from colic by the fact that pressure increases the pain, the pain is more diffuse, the abdomen is not so often rigid, it does not move on respiration, the temperature is commonly a little raised, the pulse and expression are more characteristic of abdominal disease, and the vomiting is more severe. But as any of these symptoms may accompany colic, much caution is necessary.

Appendicitis is sometimes mistaken for colic, but in this malady a little care will usually prevent a mistake.

There are other conditions, which may be confounded with intestinal colic, in which an error is of less importance, for a purgative will do no great harm; it must be remembered, however, that apart from lead poisoning, severe intestinal colic is in adults a much rarer condition than is commonly supposed. In numbers of cases of renal colic the real cause of the pain is overlooked because it closely resembles that of intestinal colic, which, again, may likewise be closely simulated by that due to a stone in the cystic duct. If the stomach be distended with gas which it cannot eject, the pylorus and cardiac opening being presumably closed, the pain produced may be very like that of intestinal colic. I have also known that form of gastric pain which comes on some hours after food, and is relieved by an alkali, to simulate colic. In exceptional cases other gastric pains may give rise to difficulty; and the possibility of neuralgia of the dorso-lumbar nerves, of disease of the abdominal muscles, of spinal disease or of locomotor ataxy, must be borne in mind.

There are many conditions of the intestine which induce pain very like the colic just described; some authors would say that such conditions give rise to colic, others that the pain was colicky. It matters not which word is used, so long as it is remembered that organic disease of the intestine may be confounded with the class of cases I have been describing. For instance, in enteritis, simple non-ulcerative colitis, mucous colitis, ulcerative colitis, and intestinal malignant disease, the pain may be sudden and griping, and thus give rise to misapprehension. Ascaris lumbricoides, by reflex action, occasionally sets up paroxysmal painful attacks; and, lastly, griping pain commonly results from purgatives.

Some authors describe a rheumatic colic, and suppose that such cases are due to exposure to cold; but we have very little exact information about them, and they are certainly far from common. Ingestion of cold liquids or ices will occasionally give rise to colicky pains. It must not be forgotten that certain cases of influenza are ushered in with sudden pain in the abdomen.

We now pass on to a very difficult subject; namely, to consider how

far some cases of colic may be called functional and fall under the heading of neuralgia or hysteria. It must be borne in mind that the sensitiveness of the intestinal mucous membrane varies very much in different persons: and, as I have said, some articles of food, which to a strong, healthy man would be innocuous, might in a weak, sickly woman act as irritants and give rise to colic. We must also remember that the abdominal organs are much under the influence of nervous conditions. For instance, hysterical vomiting is not uncommon. Numbers of kidneys are exposed by surgeons under the impression that a stone is present and none is found; and in one case, the operation having proved fatal, no stone could be discovered at the post-mortem examination. Fright will produce diarrhœa, and lunatics often complain of painful intra-abdominal sensations. There is then a considerable probability that some cases of colic may fall under the heading of neuralgia or hysteria; but it must never be forgotten that such a diagnosis should not be final until every attempt has been made to exclude it [*vide* p. 480].

The most troublesome hysterical cases are those in which there is intense pain, rigidity of the abdominal muscles, and superficial cutaneous tenderness. In these cases the resemblance to peritonitis is very close, but it will usually be found that deep pressure is not more painful than superficial, that pressure is not objected to if the patient's attention is distracted, and that other hysterical symptoms are present.

In nervous women pain closely resembling colic may be due to disease of the pelvic organs.

Many authors have suggested that in cases of colic organic disease of the intra-abdominal ganglia and sympathetic nerves might exist: there is, however, no evidence of this. I have had patients under my care in whom the abdominal ganglia were invaded by growth, but they had had no colic. I have elsewhere shown that in the human adult the semilunar ganglia are extremely variable, both macroscopically and microscopically. Of the morbid anatomy of the intra-abdominal sympathetic fibres we know nothing.

W. HALE WHITE.

DIARRHŒA

THE name "diarrhœa" is usually employed, and properly so, to indicate a fluid and too frequent discharge from the bowels. Although there may be three or four evacuations in the day, yet the term "diarrhœa" is not usually applied to them if they be properly formed and not liquid. Sometimes, however, when patients complain of "diarrhœa" we find, upon inquiry, that the motions are formed, but that there is a desire to go too frequently to the closet. Constipation is the commonest ailment of civilised countries, but diarrhœa destroys more lives than any other disease. The name "diarrhœa," like that of "constipation" or of "dyspepsia," simply denotes a symptom which may depend upon many different causes.

Causes of diarrhœa.—Diarrhœa is due to an unusually watery condition of the intestinal contents at the time when they reach the rectum, and are evacuated. This watery condition may depend upon:—

(A) Diminished absorption of water by the intestine; (B) increased secretion of fluid from the intestinal mucous membrane; or (C) on both.

Diminished absorption is the commoner cause; and it may be due to the intestine having (*a*) less absorbing power, or (*b*) less time in which to absorb. The contents of the small intestine when they reach the cæcum are normally quite fluid; but during their sojourn in the large intestine the watery parts become absorbed to a greater or less extent; so that generally they become firm, and before they reach the rectum are moulded into cylindrical masses. Sometimes, indeed, absorption goes further, and they become not only firm, but dry and hard. It is obvious that conditions which affect the small intestine alone will hardly cause diarrhœa. An irritable or catarrhal condition of the small intestine may exist, and its contents may be hurried on with great rapidity into the large intestine, yet, if the peristaltic movements of the cæcum and colon are not increased, the whole of the fluid parts of the fæces may be absorbed, and the motions may be natural or even constipated.

On the other hand, any irritation of the large intestine which results in increased rapidity of its peristaltic movements will tend to produce diarrhœa, by hurrying on the liquid fæces from the cæcum to the anus so quickly that too little time is afforded for the absorption of the watery constituents. If both the small and the large intestine have their peristaltic movements increased, diarrhœa will of course be greater than if either were affected alone.

Forms of diarrhœa.—*Diarrhœa from purgatives.*—A temporary diarrhœa may be induced by the administration of purgatives. Many of these—like colocynth, senna, cascara, castor oil, and croton oil—affect the muscular coats of the intestine, and especially of the colon; while they have little or no effect upon the processes of absorption or secretion.

They simply quicken the peristaltic movements and hurry the fluids which are poured into the cæcum by the ileum so quickly on through the large intestine that there is not time for their watery constituents to become absorbed.

Other medicines, again, especially neutral salts, greatly increase the secretion of fluid from the intestinal mucous membrane. This is shown by the fact that if three loops of intestine be isolated by means of ligatures, and a solution of a neutral salt be injected into the middle loop, and the whole be returned to the abdomen of the animal, secretion takes place to a large extent into the middle loop; so that, if the animal be killed some hours afterwards, the quantity of fluid contained in this loop will be found greatly exceeding the saline solution injected into it, while the loops on each side of it remain empty. This experiment shows conclusively that a large secretion of fluid has taken place from the intestinal mucous membrane under the stimulation of the saline solution. Some salines, however, while producing a large secretion of fluid, hardly stimulate peristaltic action at all, and thus have no purgative effect. For example, we know that acid tartrate of potash injected into a loop of intestine will cause secretion; and in a patient who has taken a dose of this medicine secretion probably takes place abundantly in the small intestine, as shown by the gurgling and rumbling in the bowels; yet the salt stimulates the peristaltic movements of the large intestine so slightly that the whole of the fluid which has been rumbling about may be absorbed, and the subsequent motion, instead of being loose, may be somewhat constipated. Accordingly, when we wish to sweep out the intestinal contents thoroughly, we combine drugs which will act upon the mucous membrane with those which will stimulate peristaltic movements; we employ, for example, the compound jalap powder, in which the acid tartrate of potash is intended to produce watery secretion, the jalap to stimulate the peristaltic movements, and the ginger to act as a carminative and prevent griping.

Diarrhœa from food.—An action upon the intestinal secretion, and movements similar to that which is caused voluntarily by means of purgative medicines, are frequently produced involuntarily by various substances taken as food, or by the products of their digestion or decomposition in the stomach and intestines. Thus, a meal too abundant in quantity, instead of being vomited, as described under Dyspepsia, may pass through the pyloric orifice, and by irritating the intestines give rise to several watery discharges: after this, the intestinal canal, being freed from its incubus, may return to its normal condition.

At other times the products of an imperfect digestion may act as purgatives. Thus fatty acids formed in the stomach, instead of producing vomiting, may cause purging; and too long a delay in the stomach will probably render laxative even such substances as milk; for we find that milk which has been artificially over-peptonised, and thereby rendered bitter, frequently causes looseness of the bowels in patients who take it. Other articles of food may contain definite poisons; for example, certain

specimens of cheese contain a toxamine, termed by its discoverer, Professor Vaughan, "tyrotoxicon." Poisonous mushrooms also contain a poison, muscarine, which causes vomiting and purging; and even esculent mushrooms contain a substance called amanitine, which, although harmless in itself, occasionally undergoes change and yields the poisonous muscarine; thus now and again violent vomiting and purging may result from the use of mushrooms which are apparently good.

Diarrhœa from microbic poisons.—Within the last few years it has been shown that albuminous substances, during the process of decomposition by microbes of various kinds, yield many poisons, amongst which are muscarine and other allied substances having a similar action; so that proteid substances, which have been imperfectly digested and have undergone putrefaction in the intestine, may be converted into powerful poisons, and give rise to violent diarrhœa.

Cholera and dysentery, enteric fever and tubercle, are all due to the action of microbes; a certain kind of dysentery being especially connected with an animal of low organisation, the amœba coli (*vide* p. 754): cholera is due to a vegetable organism or organisms, the best known of which is the comma bacillus of Koch; although Dr. Douglas Cunningham has found that a number of other species of bacillus may cause choleraic symptoms. In cholera the epithelium of the mucous membrane separates readily, though no ulcers form; in the other three diseases, however, ulceration of the intestines exists, and either causes or helps to cause the diarrhœa which is their prominent symptom. For a description of the symptoms and pathology of these diseases the reader is referred to the respective articles in this work.

The diarrhœa of tropical countries is in all probability dependent upon micro-organisms, although the exact nature of these has not been as yet determined (*vide* "Sprue," p. 774).

Fæces contain an immense number of micro-organisms; Suksdorff has estimated the number of bacteria passed daily in the stools of one person as amounting on an average to 53,124,000,000. No wonder, then, that when fæces are retained too long in the intestine they may act as irritants, and that scybalous masses may frequently give rise to diarrhœa, which ceases on their removal.

Diarrhœa from entozoa.—Entozoa present in the intestine, such as tapeworms, round worms, or threadworms, may sometimes give rise to diarrhœa. This may be due partly to the mechanical irritation; but in the case of the tapeworm it seems to be partly due also to irritant substances secreted by the parasite.

Uræmic diarrhœa.—Various poisons which circulate in the blood may be eliminated by the mucous membrane of the intestine in the same way as already described in the case of the stomach; and as in the latter organ they may give rise to vomiting, so in the intestine they may occasion diarrhœa. It is probably to the elimination of toxalbumins through the intestinal mucous membrane that the persistent diarrhœa in cases of kidney failure and threatened uræmia is due.

Diarrhœa from chills.—There can be no doubt whatever that exposure to cold, and especially to cold affecting the abdominal walls, will give rise to diarrhœa; but the mode of this action is not yet precisely ascertained. Probably the chill gives rise to an acute catarrh in the intestinal mucous membrane of the patient, just as it would cause catarrh of the respiratory passages in others; but we cannot say with certainty how far the diarrhœa is due to changes in the nervous system, in the circulation, or to tissue metamorphosis giving rise to purgative products.

Nervous diarrhœa.—Mental emotion has a powerful effect upon the intestines, so that fright frequently causes defæcation, and if continued may give rise to diarrhœa. There is a certain form of nervous diarrhœa which is often very troublesome: so long as the patient is within easy reach of a closet, and can empty his bowels whenever he feels the desire, he is perfectly well; but the moment he becomes aware that there may be difficulty or delay in evacuating his bowels, he feels the desire to do so.

Malarial diarrhœa.—This is probably to be reckoned as a form of nervous diarrhœa. In some patients it recurs regularly at the same hour of the day, resists all ordinary astringents, but yields readily to the administration of quinine.

Morning diarrhœa.—An obstinate form of diarrhœa is sometimes met with in connection with irritability of the sigmoid flexures, and sometimes also with dilatation of the stomach; it is very often known as "morning diarrhœa." The patient has to get up very early in the morning, and has perhaps three to six movements before 11 A.M. After this he is pretty free, and probably after midday he has no action at all until next morning. This form of diarrhœa is sometimes not so severe, and occurs only in the shape of one movement, just after breakfast, followed by one or two loose motions within the next hour or so, but both the severe form and the mild are apt to resist medical treatment. Morning diarrhœa is also common in persons addicted to alcohol.

Diarrhœa due to a prolapse of the sigmoid flexure into the rectum.—This diarrhœa is chronic and irritating, the motions are small, but they are numerous and often accompanied with tenesmus. The rectum in the adult is pretty firmly fixed to the bones of the pelvis, especially to the sacrum; so that, although prolapse of the rectum through the anus is common in children, it is by no means common in adults. But prolapse of the sigmoid flexure into the rectum is by no means uncommon in adults; sometimes it gives rise to constipation, but frequently it causes chronic diarrhœa. My attention was very strongly drawn to this disorder by a case that I saw some years ago in a tea-planter who had come from Assam. He had suffered from chronic diarrhœa for from six months to a year before I saw him. The whole of his trouble dated from an attack of violent constipation which had lasted many days. To relieve this he took a strong purgative, and from the moment that this acted he had suffered from diarrhœa. All the remedies he had tried had failed. On examination by the bowel I found a state of things which simulated

a prolapsed rectum in a child; but in this case the prolapse was as far up the rectum as the finger could reach. High up in the rectum I found pendent bags of mucous membrane, and in the middle of these bags I could feel the opening into the sigmoid flexure.

Malignant Disease.—To one other cause of diarrhœa I must draw attention, and that is malignant disease of the bowel. The operation of examining the bowel is a disagreeable one both for the operator and for the patient; and for this reason it is frequently omitted: but in all cases of chronic diarrhœa it ought to be done. Its omission may prove very disadvantageous to the patient, in whom malignant disease of the rectum may progress undetected until all chance of saving life by surgical treatment has been lost, and the patient is in imminent danger of death from obstruction.

Symptoms.—The symptoms of diarrhœa depend very much on the part of the bowel affected, and the extent and nature of the affection. I have already said that irritation or catarrh of the small intestine does not necessarily lead to diarrhœa; but that if it be associated with an irritable condition of the large intestine, so that the contents of the duodenum are rapidly swept down through the whole intestinal tube, the fæces are not unlikely to contain a certain amount of altered bile which either gives a green colour to the fæcal masses, or betrays itself in a yellow or greenish tinge of the mucus which accompanies the motion (*vide* "Diarrhœa of Children," p. 746).

In cases of obstruction of the common bile-duct the motions may be perfectly colourless. In such a case the urine and conjunctiva are usually deeply tinged with bile; but if the pancreatic duct alone be obstructed the motions may still be perfectly colourless, and yet no jaundice be present, nor the urine be tinged with bile. In such cases, notwithstanding their pale colour, the fæces actually contain bile; but their colour is obscured by undigested fat which usually comes from the milk or cream in the diet. In sprue (p. 779) the motions are generally frothy.

In some cases, owing to decomposition of the intestinal contents, the fæces have a most abominable smell. The exact nature of the substances to which these smells are due has not been determined; but similar substances are readily produced by digesting fibrin with active pancreas at the temperature of the body for about eighteen hours.

When the diarrhœa is severe, the discharges excessive, and the motions abundant and watery, the blood becomes drained of its watery parts, circulation becomes feeble, there is a tendency to venous stagnation, and signs of general collapse may appear. These are most marked in cases of Asiatic cholera (vol. i. p. 898), but may be noticed also to a slighter extent in patients suffering from ordinary diarrhœa, and especially in children. The pulse is small and feeble, the skin pale and cold, the face shrunken, the eyes sunken in their orbits; cramps may attack the legs; and in children, on account of the lowered blood pressure, the fontanelle is greatly depressed. On account of the frequent passage of

the motions great tenderness, soreness, and excoriation of the anus, and even of the nates, may occur.

The irritation which gives rise to diarrhœa is frequently accompanied by pain, and this may be so excessive as to lead to faintness or actual syncope, especially in persons who suffer from a feeble heart. Even when pain is not present diarrhœa is apt to produce or be accompanied by malaise, languor, irritability sometimes, and, if excessive, by general prostration.

Chronic diarrhœa dependent upon local irritation in the rectum may go on for a long time without any indication whatever of general depression, emaciation, or disturbance of any kind, beyond the simple annoyance which is caused to the patient by repeated calls to the closet. In cases where much mucus is passed, and especially, I think, in those cases where the sigmoid is prolapsed into the rectum, a condition of hypochondriasis is very frequently observed. In any case there is great depression of spirits and inability to take any interest in external things; the attention of the patient is concentrated upon his own condition, and more especially upon the state of his bowels.

In many cases of this sort mucus is passed in large quantities, either surrounding the motion or separately; and sometimes the amount is so great that it appears to form a regular cast of the bowel, and has been described as membranous enteritis. Two varieties of mucous diarrhœa have been described as membranous enteritis and mucous colic. In the first of these there are recurrent spasmodic pains; in the second there is more generally a feeling of continued discomfort and uneasiness, frequently accompanied by a tendency to nervous depression and hypochondriasis. The causation of these conditions has not been precisely ascertained; and I am inclined to believe that in many cases they depend upon prolapse of the rectum into the sigmoid.

In cases such as I have described there is generally a sense of incomplete evacuation of the bowels, even soon after the motion has passed; and the more the patient strains the worse this feeling is apt to become, because the prolapsed part of the bowel becomes more congested.

Ulceration of the rectum gives rise to diarrhœa, and also sometimes to a desire to sit long at the closet. I am inclined to think that such ulceration occasionally occurs from mechanical injury to the mucous membrane of the bowel by the bone or ivory nozzles of enema syringes; to avoid this it is better to use a soft india-rubber tube, which is not likely to hurt the bowel.

In some cases there is a constant desire to defæcate, although there be nothing whatever present in the bowel, and nothing can be felt wrong with its wall. I have seen one case in which the desire was so incessant that the patient, an old woman, sat permanently on a bedpan: the only opinion that I could form was that the eczema, from which she suffered, had affected the mucous membrane of the intestine, and gave rise to a constant irritation in the rectum similar to that which it caused in the skin.

Usually diarrhœa is either accompanied or preceded by intestinal pain; but this is not invariably the case. This pain sometimes occurs in the small intestine, but more usually in the colon; hence its name of colic. Like those other pains—such as hepatic and renal colic—to which the name of colic has been extended, which also occur in organs containing involuntary muscular fibre, the pain in the intestine probably depends upon irritation to the intestinal nerves, partly by cramp-like contractions of the involuntary muscular fibre in some sections of the bowel, partly by excessive dilatation in other sections. Such pain may occur without diarrhœa, because the cramp-like contractions tend rather to interfere with the passage of the intestinal contents than to accelerate them; in lead colic the pain is very marked, but is usually accompanied by obstinate constipation.

In migraine the pain in the head is associated either with excessive contraction or excessive dilatation of the arteries, and, sometimes at least, with dilatation of the proximal and contraction of the distal ends of the arteries supplying that part of the head in which the pain is felt. In this ailment there seems to be at the same time a dilatation of the proximal, and a contraction of the distal, end of the stomach, the fundus being much dilated and the pylorus firmly contracted. It is probable that a like condition occurs to some extent in the intestine, and either in consequence of this, or of contraction of the intestinal vessels, similar to what occurs in the branches of the carotid in migraine, a pain comes on in the intestine similar to that of headache. In some persons the two come on together; but in others the pain appears sometimes in the head and sometimes in the stomach, the stomach being free when the head is painful, and conversely. In certain cases diarrhœa occurs along with headache, and appears to replace the vomiting which usually accompanies migraine.

Treatment.—The treatment of diarrhœa consists, first, in removing any irritant which may be giving rise to it; and, secondly, in soothing any irritation of the intestine itself which may remain after the irritant has been removed. In the case of artificial diarrhœa, due to purgatives, great care must be taken to ensure their removal; and thus it is the common practice to give a saline purgative next morning after a dose of calomel. This practice is not only useful in ensuring the removal of waste products from the intestine, but of clearing out the calomel itself; sometimes if the calomel be taken alone at night, and no saline in the morning, the calomel will act as a powerful irritant, giving rise to much pain: in one case which I saw it nearly produced collapse, very much as if a dose of corrosive sublimate had been taken; and similar results sometimes arise from a colocynth pill or from proprietary pills.

In the case of diarrhœa caused by food, either excessive in quantity, objectionable in quality, or so decomposed in the intestine as to be irritating, one of the best means of stopping the diarrhœa is to give a dose of castor oil together with a little opium—as for example, half an ounce of castor oil and 8 minims of tincture of laudanum. The castor oil clears out the intestine thoroughly, irritant matters are removed, and the opium

soothes the bowel and allows it to recover from the irritation. If, in place of a purgative an astringent be given, the movements of the bowels are temporarily arrested, but the irritant remains there; and, if it consist of decomposing food, its retention makes matters worse, so that after a day or two the diarrhœa returns in a severer form than before.

Another favourite remedy for diarrhœa due to indiscretions in diet is compound rhubarb powder; the rhubarb and magnesia act as purgatives and clear out the intestine, while the slight amount of tannic acid contained in the rhubarb acts subsequently as an astringent.

When the diarrhœa is due to irritation in the mucous membrane of the intestine itself, as in the catarrh which succeeds violent irritation and not to irritating substances lying within the bowel, one of the most common remedies is chalk mixture. Lime forms with fatty acids an insoluble soap, and will thus neutralise such acids, which are strong intestinal irritants. It will also neutralise other acids not of the fatty series, and by its combination, either with acids or albumin, will tend to form a non-irritating pellicle over the mucous membrane; while at the same time lime salts slow the movement of involuntary muscular fibre, and tend to lessen peristalsis. At the same time the carminatives present in the chalk mixture aid the expulsion of flatus, and prevent the pain which would otherwise arise from its distending the bowel. When the pain is severe, and chalk mixture alone is insufficient, its combination with opium in the form of the pulvis cretæ aromaticus cum opio of the Pharmacopœia is exceedingly useful; or 5 to 10 minims of tinctura opii may be added to the ordinary chalk mixture. It is hard to explain the effect of brandy in checking simple diarrhœa; but there can be no doubt that it is a most useful remedy, and an ounce of neat brandy is a most useful remedy in simple diarrhœa. Its efficacy may be increased by the addition of 5 to 10 minims of tincture of opium, or 20 minims of chlorodyne: these remedies indeed often prove very useful in checking diarrhœa without brandy, or anything else. It must be borne in mind, however, that if the diarrhœa depend upon irritating substances, the use of these remedies, while giving relief for a time, may render the diarrhœa more troublesome if they are taken before the irritant substances have been passed out. If, however, the diarrhœa have continued for some days and the irritant be already removed, the brandy and opium may soothe the intestine and effect a permanent cure.

In many cases diarrhœa is due to mixed causes, and there are both irritating substances in the intestine and an irritated or inflamed condition of the mucous membrane itself. A useful plan of treatment is, therefore, to give, first, a dose of half an ounce of castor oil with 5 to 10 minims of laudanum. An hour afterwards let the patient take some warm tea or other warm drink to assist the oil. After it has acted freely administer a sedative such as the following:—℞ Bismuthi carbonatis, Sodii bicarbonatis, āā gr. x.; Spt. chloroform, ♏ x.; Aq. menthæ piperitæ, vel Aq. cinnamomi, vel Aq. caryophylli, ad ℥j. Ft. Hst. This may be given every four hours twenty minutes before food. At the same time

an ounce of mistura cretæ, to which 5 to 8 minims of laudanum may be added, should be given after every loose motion. This direction regulates the amount of astringent which the patient takes according to his requirements; for if the bowels are only opened once loosely he takes but one dose, whereas if there are ten loose motions he gets ten doses.

For patients who are travelling, or who cannot stay at home on account of business engagements, draughts are awkward; for them bismuth lozenges, soda-mint tablets, or 20-grain powders of pulvis cretæ aromaticus, or pulv. cret. aromat. c. opio, are most convenient.

In place of giving opium by the mouth the drug may frequently be administered with great advantage by the bowel; either as the ordinary enema opii containing half a drachm of laudanum in two ounces of starch mucilage, in the form of a morphine suppository, or of a compound lead suppository, where the astringent action of the lead tends to supplement the soothing action of the morphine.

The pain which frequently accompanies diarrhœa may be greatly lessened by the application of a large warm fomentation or poultice to the abdomen. In cases where these remedies cannot readily be applied, as on a railway journey, relief may be obtained by applying a tin or india-rubber bag containing hot water to the abdomen over the clothes. In the case of a poultice it is best not to apply it, as is sometimes done, directly to the skin, or with only a thin bit of muslin between it and the skin, for in this fashion the poultice must either be allowed to become half cold or the patient's skin will be scalded. Much more relief is afforded by putting two folds of flannel between the skin and the poultice, which may then be made boiling hot, and the heat, coming gradually through the flannel, does not burn the skin.

Carminatives frequently lessen the pain greatly by rendering the contraction of the bowel more even, lessening spasm, and assisting the escape of flatulence. A useful mixture is half a drachm of aromatic spirit of ammonia, half a drachm of compound tincture of cardamoms, and ten or fifteen minims of spirit of chloroform. Ten minims of compound spirit of ether may be substituted for the spirit of chloroform in the mixture or added to it; and if the pain be very great, half a drachm to a drachm of the compound tincture of camphor makes a useful addition. The whole of this may be given in an ounce of water, or, still better, perhaps, in peppermint water.

In cases where distension of the colon with flatus is very great, it may sometimes be relieved by passing a long flexible india-rubber tube into the colon; but usually the wind may be brought away by injecting about four ounces of dill-water, pepper and water, or some other carminative. Or an enema of turpentine may be employed; but probably the best of all injections for the relief of tympanitic distension is the enema assafœtidæ, although its disagreeable odour prevents it from being used so frequently as, from its great power of relieving flatulence, it would otherwise be.

Diarrhœa due to decomposed food, to mushrooms, or to cheese, is

best treated by clearing out the bowels by castor oil; but along with this belladonna or atropine should be given, because atropine is an antidote to muscarine, the poisonous alkaloid of mushrooms, and to a substance which is likewise formed by the decomposition of albumin. Ten minims of the tincture of belladonna, or $\frac{1}{100}$th of a grain of atropine, may be given every half hour, or every hour, either until the diarrhœa is less, or till the symptoms of physiological action have begun to show themselves in dryness of the mouth or dilatation of the pupil. Of course this remedy must be used under constant supervision, as it is hardly safe to leave it to the patient's friends to decide when the drug is to be stopped.

In a case where diarrhœa is due to microbic infection one may combine the administration of various antiseptics, to destroy the microbes themselves, with measures for the removal of the poisons which they form. Among the most powerful of these is mercury in various forms—gray powder, calomel, corrosive sublimate, and periodide.

The first two may be given in single doses of five grains of gray powder, or two to three of calomel; or in divided doses, such as one-third to two grains of gray powder, and one-third to one-half a grain of calomel, every two to four hours. One thirty-second to one-sixteenth of a grain of periodide or of corrosive sublimate, or half a drachm to a drachm of the liquor hydrargyri perchloridi, may be used in a similar way.

Bismuth, in the form of carbonate, subnitrate, or salicylate, is a useful intestinal sedative; the carbonate is the form which acts best with chalk, but the salicylate is the most powerful disinfectant salt of the three, and may be given with salicylate of soda or with salol. Salol passes through the stomach unchanged, but becomes converted in the intestine into salicylic acid and phenol, both of which are powerful disinfectants. It may be given in doses of ten or fifteen grains in a cachet, alone or with salicylate of bismuth. β-Naphthol and its compound with salicylic acid, called naphthalol or betol, are both used (in doses of 5 to 10 grains) in the same way, and for the same purposes, as salol. Naphthaline is another useful intestinal disinfectant, and is one of the most powerful of them all in deodorising offensive fæcal motions. In the diarrhœa of children, for example, it completely removes the offensive odour, but, so far as my own experience goes, it will not by itself arrest the diarrhœa; it has been stated by others, however, to do so.

Besides killing the pathogenetic bacteria in the intestine by means of intestinal disinfectants, they may be destroyed by the simple plan of starving them out. Although MacFadyen and I found that bacteria appeared to have the power of adapting themselves to changed foods, and of manufacturing new ferments wherewith to digest them, yet a certain time is required for this adaptation; and if the food be rapidly and frequently changed the bacteria may be starved. Thus, when milk has been used as a diet, and the intestine has become infected with bacteria which live readily upon milk, the complete stoppage of the milk for a while, and substitution for it of farinaceous food only, will tend to kill off

a large number of the bacteria which thrive on milk. After a day or two other bacteria may grow and multiply upon farinaceous food; but if this again be suddenly changed for a meat juice, a great number of these will be killed off, and then by reverting to milk, and so on, the various bacteria may be gradually eliminated (*vide* also articles on "Cancer," "Cholera," "Diarrhœa of Children," "Dysentery," "Enteric Fever," "Phthisis," "Sprue," "Tuberculosis").

In the cases of ordinary diarrhœa, which are so common in persons who have been living in India, Afghanistan, or other tropical climates, medicines are frequently useless; and the only method of cure is that, proposed by Sir Joseph Fayrer, of putting the patient on an absolutely milk diet with no other food whatever; the patient, in fact, is treated exactly as if he had typhoid fever. In some cases the patient may be able to attend to his work and even lay on flesh, but this is not usually the case; and, as a rule, while on the milk diet he will have to be kept in bed.

Astringents of various kinds are frequently useful in diarrhœa after the acute stage has passed off, and it has become chronic. The chief astringents are some of the heavy metals in various forms. Rhubarb, as already mentioned, is useful, as after clearing away irritants from the intestine its rheo-tannic acid has an astringent action. Catechu, in various forms, rhatany, logwood, are all occasionally useful. The more soluble preparations, such as infusion and tincture of catechu, expend part of their energy on the stomach before passing into the intestine; and therefore the compound catechu powder, in which the three astringents, catechu, kino, and rhatany, pass into the intestine along with two aromatics, cinnamon and nutmeg, is perhaps more useful, in doses of twenty or thirty grains. Compound kino powder in 10 grain doses is also very useful, the kino acting as an astringent, and the opium lessening pain and irritation. In a very chronic case of diarrhœa the lead and opium pill is useful, and so are the sulphates of zinc and copper in doses of one to two grains in the form of pill, either alone or with opium.

In all cases of chronic diarrhœa the urine should be carefully examined, and if it be found to have a persistently low specific gravity, and to contain a trace of albumin, great care should be taken not to check the diarrhœa hastily by means of opium or astringents; in such cases the bowels form the chief channel for elimination of the products of tissue waste, and if the diarrhœa be checked the patient may quickly die of uræmia.

In cases of tendency to diarrhœa from cold, and, indeed, in all cases of chronic diarrhœa or tendency to diarrhœa, the abdomen should be kept at a warm and equable temperature, either by a flannel bandage or by a silk scarf wound several times round it.

Attention should also be paid to the feet, the shins, and the back of the neck, as a chill in those spots is not unlikely to recall the diarrhœa or abdominal pain, even although the abdomen itself be kept warm.

Cases of nervous diarrhœa are to be treated by moral remedies; the patient should be encouraged to resist the inclination to defæcate, just as a patient with a nervous irritability of the bladder is told to resist the desire to micturate.

In one particular class of nervous diarrhœa, namely, that where the introduction of food into the stomach gives immediate desire to go to stool, bismuth before meals tends to check the impulse; the same result is sometimes obtained, even more easily, by the use of liquor arsenicalis, in doses of half a minim to a minim in an ounce of water or peppermint water, ten or fifteen minutes before meals. In cases of diarrhœa depending upon locomotor ataxy small doses of antipyrin may prove useful.

Malarial diarrhœa, although it resists the usual astringents, often yields readily to quinine or cinchona. When in such cases the liver is much enlarged and tender, the cure may be quickened by the administration of a mercurial followed by a saline purgative; thus the congestion of the liver is reduced before quinine or astringents come into the field.

Morning diarrhœa may generally be greatly relieved and sometimes entirely checked by the simple plan of telling the patient to take no liquid after five o'clock in the afternoon; all the liquids should be taken in the early part of the day. In persons, on the contrary, who suffer from evening diarrhœa, the liquids should be taken late at night, and very little during the early part of the day. In all cases the use of alcohol should be carefully regulated.

In cases where diarrhœa depends upon prolapse of the sigmoid flexure into the rectum, it should be treated by avoiding all articles of food which would be likely to pass through the intestine undigested and to irritate the tender part of the bowel. The bowels may be kept open by means of an enema of half a pint of water every morning; and immediately after the action an astringent injection of one or two drachms of catechu in two ounces of water should be thrown into the bowel and retained there. In place of the injection, ointment may be introduced by means of one of Allingham's ointment carriers. If there be irritation in the sigmoid flexure itself, I have had an ointment carrier, similar to Allingham's, made of a larger size; if a soft india-rubber tube be attached to it the ointment can be passed well up into the sigmoid.

In cases of very chronic diarrhœa, more especially where dysenteric or other ulcers are supposed to be present, large clysters of disinfectant and astringent solutions are sometimes useful. For this purpose a pint or more of the solution should be employed in the manner already directed for the softening of scybala. These clysters may consist either simply of warm water or of thin starch paste, to which are added various sedative and astringent substances, such as carbonate of bismuth (5 to 10 grains per ounce), boric acid (1 grain per ounce), salicylic acid ($1\frac{1}{2}$ grains per ounce), thymol ($\frac{1}{8}$ to $\frac{1}{4}$ of a grain per ounce), nitrate of silver, sulphate of copper and chloride of zinc ($\frac{1}{2}$ grain to 3 grains per ounce). The clyster should be retained as long as it can be with comfort. On

account of the risk arising from absorption, powerful poisons like corrosive sublimate, iodide of mercury, or carbolic acid are inadmissible.

In cases of malignant disease of the bowel relief may be afforded by a sedative suppository such as the following:—℞ Ext. belladon. gr. ½, Morphinæ gr. ⅓, Cocainæ gr. ½-1 gr., Ol. theobrom. *q.s.* Ft. suppositorium. If the diseased part of the bowel cannot be removed by surgical interference, the bowel should be kept open by the use of a warm water enema thrown up the bowel high enough to pass beyond the obstruction and soften the fæces above; so as to prevent any hardened mass coming down upon the constricted portion and thus giving rise to sudden obstruction. When obstruction threatens an artificial anus must be made; but when diarrhœa is due to malignant disease of the descending colon or of the sigmoid, relief may be afforded by opening the abdomen and connecting the bowel above and below the diseased portion, so that the fæcal matters pass by the affected part instead of through it.

The operation of complete excision of the diseased part with union of the ends of the intestine has not been very successful hitherto; but if surgery improves as much within the next quarter of a century as it has done in the last, it is possible that such an operation may be frequently and successfully performed, and patients cured in cases and under conditions at present regarded as hopeless.

T. LAUDER BRUNTON.

REFERENCES

1. BRIEGER. "Zur Kenntniss du Fäulniss-alkaloide," *Zeit. für phys. Chemie*, Bd. vii. p. 274; "Ueber Spaltungsprodukte der Bacterien," *Ibid.* Bd. viii. p. 306; "Zur Darstellung der Aetherschwefel säure aus dem Urin," *Ibid.* Bd. viii. p. 311; "Ueber Spaltungsprodukte der Bacterien," *Ibid.* Bd. ix. p. 1.—2. BRUNTON, LAUDER. "Action of Purgative Medicines," *Practitioner*, 1874, vol. xii.; "On some Forms of Diarrhœa, especially Morning Diarrhœa," *Quarterly Medical Journal*, Jan. 1894; and various papers collected in *Disorders of Digestion.*—3. BRUNTON, LAUDER, and PYE-SMITH, *British Association Reports*, 1874, 1875, and 1876; and *Practitioner*, vol. xxxiii. pp. 353, 412; vol. xxxiv. pp. 96, 196, 265, 350, 418.—4. CHAUVET, A. "Diarrhée matinale," *Thèse de Paris*, 1888.—5. FAYRER. "Chronic Diarrhœa of India and the Tropics," *Lancet*, 1876, vol. ii. p. 389; "Liver Abscess and Dysentery," *Ibid.* 1881, vol. i. p. 803; "Tropical Diarrhœa," *Ibid.* 1889, vol. i. p. 734.—6. MOREAU. "Action du sulphate de magnésie sur l'intestin," *Arch. gén. de méd.* Aug. 1870, p. 234.—7. MUSSEY, GUENEAU DE. *Clinique médicale.* Paris, 1874.—8 NOTHNAGEL. *Erkrankungen des Darms*, Wien. 1895.—9. RADZIEJEWSKI. "Zur physiologischen Wirkung der Abführmittel," *Arch. für Anat. v. Phys.* 1870, p. 95.—10. SCHMIEDEBERG and KOPPE. *Das Muscarin.*—11. SUCKSDORFF. "Das quantitative Vorkommen von Spaltpilzen im menschlichen Darmkanale," *Arch. für Hygiene*, Bd. iv. 1886, p. 355.—12. VULPIAN. "Sur l'action des purgatifs salins," *Bull. gén. de Thérapeut.* 1873, vol. i p. 522.—13. VAUGHAN. "Ein Ptomaine aus giftigen Käse," *Zeit. für phys. Chemie*, Bd. x. 146.

T. L. B.

The Diarrhœas of Children

In the young child—especially in the infant with his feeble hold upon life—a looseness of the bowels may be a matter of no little moment. It is, indeed, to this derangement that a large number of the deaths which occur in infancy are due. The growing child is more dependent than the adult upon a continuous supply of food; for, with a poorer blood, he has to carry on a more considerable work. The adult has only to maintain the current nutrition of the body; the child, while doing this, has also to provide material for growth and development. His organs, therefore, are always kept hard at work; and that they may work to good purpose, the blood must be maintained at a normal standard by a regular inflow of nutritive material. When the supply is suddenly interrupted, as must happen in any derangement which gravely affects the digestive functions, the blood at once becomes poor and nutrition flags. If to this state of oligæmia be added the exhaustion induced by a copious drain from the bowels, the consequences to a young infant may be disastrous.

Diarrhœa is a symptom, and as such may be met with in every degree of severity from a simple looseness of the evacuations, which causes little reduction of strength and yields readily to treatment, to a profuse watery drain which exhausts the tissues of fluid and leads to death in a few hours. The age of the patient, the state of his general health, the cleanliness of his surroundings, the cause or causes to which his illness is owing, and the pathological conditions which accompany it, are all matters which may have a very definite influence upon the course and issue of the complaint.

Etiology.—In its simplest form diarrhœa is the consequence of a mild catarrh due to direct irritation of the intestinal mucous membrane, or to exposure to cold. In hand-fed babies looseness of the bowels is often excited by unwise feeding; for the food, as it passes undigested along the alimentary canal, ferments and sets up catarrh of the mucous membrane. This is especially apt to occur in nurseries where the supply of food for the whole day is prepared in the morning, as fermentative processes soon begin in the sweetened mixture. But it is not infants only who suffer in this way, for in children of all ages irritability of bowel may be caused by improper food. The looseness is, no doubt, an effort of the digestive canal to rid itself of the offending matter; and if the dietary be amended at once the stools soon recover their normal appearance.

Chilling of the surface is another cause of simple diarrhœa. Infants when first short-coated often suffer in this way. Sometimes it is in the nursery bath that the chill is contracted, for with many nurses it is a matter of principle to reduce the temperature of the bath water to the lowest point the child can bear without showing signs of distress; and in

a susceptible child a prolonged bath in water which is merely tepid will often set up the complaint.

The rapid alternations of temperature which are so often met with during an English summer—the temperature falling suddenly 10° or 15°—are almost always followed by an outbreak of diarrhœa amongst the children. Ricketty subjects, probably on account of their profuse perspirations, are especially prone to suffer from these atmospheric changes; and in teething infants the process of dentition, as it excites a certain amount of fever, is often accompanied by a mild relaxation of the bowels. In the latter case it is to the pyrexia, and not to the accidental cause of the pyrexia, that the complaint must be ascribed. The child suffers not because he is cutting a tooth but because he is feverish, and therefore keenly susceptible to changes of temperature. But even a mild purging is usually something more than a mere catarrh of the bowel. The relaxation is accompanied by fermentative processes which are due, no doubt, to the action of bacteria. Escherich found that in undigested milk discharged from the bowel two organisms were invariably present and in large numbers; namely, the bacillus coli communis and the bacterium ærogenes. These microbes attack the milk sugar especially, and a state of catarrh of the bowel seems to supply the most favourable conditions for their operation.

The same causes which set up a simple diarrhœa in a healthy child will often induce a much graver disturbance in one whose nutrition is faulty, or who is living under imperfect sanitary conditions. But the worst forms of diarrhœa occur during the summer heats, and seem to be the direct consequence of the heightened temperature aided, no doubt, by septic changes in the contents of the bowel. It has long been held that the prevalence and fatality of the summer diarrhœas have a definite relation to the degree of heat; and Dr. Dawson Williams has brought together much valuable evidence tending to show that the prime element in the etiology of the illness is a high minimum temperature. Dr. Henry Tomkins was the first to urge the value of exact observations upon the temperature of the earth, for heat which has sunk for a few feet into the ground is parted with but slowly, and a heated soil must do much to maintain the temperature of the air. He found that as soon as the thermometer registered 59° to 62° F. at a depth of one foot below the surface the causes producing the disease began to operate. Dr. Ballard pushed the inquiry to a depth of four feet, as at this distance from the surface the temperature rises and falls more slowly still. He states that in summer the mortality from bowel complaint does not begin until at four feet the thermometer records a temperature of 56° F. This statement has been confirmed by Dr. J. Priestley, who showed that the rise in the diarrhœa death-rate followed a rise in the earth temperature to 56°, and fell again as the heat of the soil sank below that point.

In its worst forms, such as occur mainly in children under two years of age, summer diarrhœa is rarely seen out of large towns, and is most virulent in seasons when great heat is combined with drought. Many

observers regard the affection as a contagious zymotic disease; and Dr. G. B. Langstaff refers its origin to sewer air, or to a miasma bred locally from the soil. The stools have been searched frequently for bacteria. Dr. Booker found more than thirty varieties of microbe; and from three of these Dr. V. C. Vaughan obtained important albuminous products which, when injected under the skin of kittens, caused vomiting and purging and, if in sufficient quantity, collapse and death. Finkelstein also has reported the discovery in the stools of abundant rod like micro-organisms, which bear a close resemblance to the B. coli communis but show certain differences by cultivation on potato. As yet no special organism has been found invariably associated with the attacks; but Dr. Booker states that a group which he calls the "proteus" group of bacteria was represented in fifteen out of nineteen cases. It is probable that milk is mainly the medium by which the germs find their way into the alimentary canal; and certainly to one who is familiar with the indifference to cleanliness commonly shown by farm labourers, and with the filthy state in which the cows often return from their pastures and stand about in their stalls, contamination of the milk ceases to be a matter for wonder. The influence of heat in such a case may be limited, perhaps, to furthering rapid development of the bacteria.

Inflammatory conditions in the cow may also have a bad effect upon the milk. Holst in four separate epidemics of acute gastro-intestinal catarrh found the streptococcus longus in milk supplied by cows who were suffering from mastitis. He states that these cocci, when injected into mice and rabbits, set up diarrhœa, and when swallowed by himself caused colic and vomiting. In serious cases the ill effects produced by the microbes are not mere fermentative processes as in the milder varieties of diarrhœa, they seem rather to depend upon decomposition of the proteid constituents of the milk and the formation of noxious chemical compounds; these when absorbed give rise to the toxic symptoms. Certainly in the more virulent cases the whole course of the illness points to a septic origin, especially as the patient often dies, not merely exhausted by the drain from the bowels, but in a state of nervous collapse, which may continue for many days after the character and number of the stools has ceased to be a cause for anxiety. This septic variety of diarrhœa is no doubt most common during the first two years of life, but it is seen also with fair frequency at a later period of childhood. It attacks strong subjects as well as weakly ones: indeed, a condition of sturdy health, although it may influence the issue of the illness (but even this is doubtful), seems to afford little protection, if any, against its onset. This cannot be said of other non-septic forms of purging, however serious they may be. As a rule weakness in the child—a condition which implies a lowered power of resisting hurtful influences—tends to favour the occurrence of inflammatory diarrhœa; and in many diseases a looseness of the bowels is a familiar complication. In typhoid fever, measles and scarlatina, diarrhœa is common: all forms of catarrhal complaints may be accompanied by it; and causes which set up congestion of the portal system, such as cirrhosis

of the liver, and diseases of the heart and lungs, lead to obstruction in the whole venous system and help to induce it.

Morbid anatomy.—The morbid appearances connected with inflammatory states of the bowel in the adult are dealt with at length elsewhere. In the child the lesions are much less constant than is the case with older persons, and it is often a matter of surprise that after death from a severe attack of bowel complaint the morbid appearances should be so ill-defined. This curious want of correspondence between the serious symptoms observed during life and the slender traces noted later in the bowel may be seen both in acute and chronic cases. Inflammatory redness and swelling, patches of false membrane, hæmorrhagic extravasations, and more or less deep ulceration of the mucous membrane may all be present; but we never feel certain that we shall find them. We see them in one case, in another which presents symptoms apparently identical we look for them in vain. Ulcerations of the bowel are more common in the subacute and chronic cases than in those which run a shorter course, and are often suspected when not actually present. In the worst forms of choleraic diarrhœa the most marked changes are seen not in the bowel but in distant parts of the body. Anæmia of the brain and other organs is common; but sometimes there is hyperæmia of the meninges and cerebral cortex due, possibly, to the action of microbes or of the poison they originate. In the chronic form of diarrhœa meningeal hæmorrhage may occur, or the dural sinuses may be blocked by thrombuses from infective phlebitis. Marfan attributes all these lesions to microbic agency which sets up toxi-infective processes, and states that chronic affections of the meninges and encephalon, such as chronic hydrocephalus and cerebral sclerosis, may arise from an attack of acute gastro-enteritis in infancy. Of other organs the kidneys are apt to be the seat of parenchymatous nephritis. Often the liver is fatty, and in cases of entero-colitis, when the illness is of some weeks' duration, this fatty degeneration is seldom absent. In addition, patches of broncho-pneumonia may be found in the lungs. Sevestre has drawn attention to this complication and attributes it to direct infection. In four out of five cases of broncho-pneumonia complicating enteritis, under the care of this physician, Lesage obtained pure cultures of the bacillus coli from the patches in the lung. The latter observer states that he has found the virulent bacillus coli in the air round the beds of children suffering from infective diarrhœa, and even in sterilised milk exposed for a time in the wards. Under such conditions the spread of the disease to children previously healthy, and the occurrence of infective broncho-pneumonia, are not difficult to explain.

ACUTE DIARRHŒA.—**Varieties.**—For convenience of description three forms of acute diarrhœa in infants and children will be described: A, simple non-inflammatory diarrhœa (mild intestinal catarrh); B, inflammatory diarrhœa (acute entero-colitis); and C, choleraic or septic diarrhœa (infantile cholera). Chronic diarrhœa will be discussed afterwards.

A. Simple non-inflammatory diarrhœa.—This variety is a mere disorder of function due to a mild intestinal catarrh. The purging yields readily to treatment if the conditions are favourable; but if it be aggravated by insanitary influences it may pass into one of the other varieties and may then prove fatal.

Symptoms.—In infants the looseness may begin quite suddenly, or be preceded for some hours by signs of indigestion and abdominal pain. The tongue is furred and the child may retch or even vomit. He is peevish and restless, and as he cries draws up his legs uneasily as if griped. At this time the temperature may be elevated a couple of degrees or so, but there is usually a fall after the first discharge from the bowels. The earlier motions consist of thin fæcal matter containing small lumps of undigested curd. They are usually sour-smelling or even frothy from fermentation. As the discharges continue they become thinner and, if the lower bowel be the part chiefly affected, they often contain visible mucus. They are then passed with some straining, and a streak of blood may perhaps be noticed on the diaper. The motions are often coloured green. This, according to Pfeiffer, is due to the conversion of the bilirubin of the bile into biliverdin, owing to an abnormal alkalinity of the contents of the bowel. In this case the stools contain a large quantity of bile, and their reaction is neutral or only faintly acid. But according to some observers there is a distinct variety of green stool which is contagious. It owes its colour, they say, to a special pigment which is generated by a rounded bacillus. Lesage even states that he has succeeded in cultivating this microbe and in setting up a green diarrhœa in animals by injecting it into the blood. This experience has not as yet been confirmed by the experiments of others; but I think many clinical observers may be disposed to agree with Lesage as to the occasional contagiousness of an apparently harmless diarrhœa.

At first the motions are passed frequently and in large quantity; but soon they become less numerous and only number five or six in the twenty-four hours. As a rule griping is not noticed after the first few dejections, and there is no tenderness or swelling of the belly. The child, however, evidently suffers in his nutrition; his colour fades, and his flesh very quickly becomes soft and flabby. This form of looseness appears to be due to direct worry of the mucous membrane, and tends to subside of itself when the irritating matter has been discharged. If in warm weather it persist beyond a very few days it may pass into a more serious form.

In older children the symptoms are much the same as in infants; but as the patient is stronger his nutrition suffers less, and although he may look pale he loses little flesh. His tongue is furred, but unless the stomach is disordered as well as the bowels the child is in good spirits and takes his food well: indeed, if not troubled with abdominal pain he will not allow that he is ill.

There is a special form of looseness of the bowels to which children are subject at the age of five or six years and upwards. It is called

"lienteric diarrhœa," and appears to be due to an exaggerated peristaltic action of the bowels, for the stools follow directly upon a meal with great urgency and are preceded by severe abdominal pain. The child may be noticed to turn pale soon after sitting down to table, and often rises and hurries from the room before the meal is at an end. The same thing occurs whenever food is taken, and, in addition, the bowels may act in the early morning, or when the child first leaves his bed. The motions consist of undigested food and mucus. Sometimes the pains are complained of without being followed by a stool; and sometimes, although the bowels act with urgency after a meal, abdominal discomfort seems to be absent. The tongue is slightly furred, or is red and irritable-looking with projecting papillæ. This form of looseness interferes greatly with nutrition. The act of eating seems to rouse the muscular coat of the bowel to excessive movement; it is not that the stools are numerous, but that they contain, little changed, nearly all the food that has been taken. The child may eat heartily enough, but the meal is carried so quickly along the alimentary canal that the process of digestion can hardly begin. As a consequence the patient soon becomes pale and languid and thin; and if the derangement continue, as it often does for weeks together, his evident loss of flesh causes the greatest anxiety amongst his friends.

B. Inflammatory diarrhœa (entero-colitis).—Inflammatory diarrhœa is a much more serious complaint than that just described. While it lasts, not only is nutrition put a stop to, but the patient's little stock of strength is reduced by high fever and a profuse watery diarrhœa. The disorder, therefore, is a serious trial even to a sturdy child, and often proves fatal to feeble children and infants. In such patients symptoms of depression come on early if the purging be severe, and, moreover, in infancy there is a special tendency to deficient action of the kidneys which adds greatly to the danger of the illness.

This form of diarrhœa is associated with evidences of inflammation, and sometimes, in protracted cases, of ulceration of the intestinal mucous membrane. It may begin suddenly or gradually, and often follows upon an attack of mild intestinal catarrh, or is a sequel of the septic form of diarrhœa to be afterwards described. When established its chief features are a high bodily temperature with frequent and watery discharges from the bowels. It is difficult to say whether all the symptoms are due to the anatomical changes noted in fatal cases, or whether unwholesome fermentations in the bowels and the absorption into the system of poisonous ptomaines may not be answerable for some of them. I believe that this noxious decomposition is present at any rate in the cases where early signs of collapse are noted, or where the course of the illness is exceptionally rapid and severe.

Symptoms.—As a rule the first symptoms are those of an intestinal catarrh. In infants there is some abdominal pain, as shown by cries and uneasy movements of the legs; and the loose stools consist of undigested and fermenting matter. If there be any gastric catarrh the child vomits

also. After the food contained in the bowels has been evacuated the stools get thinner and more offensive, and are tinted brown or green. Often they number ten, fifteen, or twenty in the day and night, and the more frequently they are passed the thinner they become. Usually they vary in character from time to time and, although always very offensive, are thin and pasty, or frothy and dark, or consist of a greenish watery fluid which deposits a small quantity of thin fæcal matter. So long as the lower bowel is unaffected there is no mucus visible to the naked eye. Under the microscope they are found to consist of particles of casein, starch granules, fragments of meat fibre, epithelial and round cells, many varieties of bacteria, and enormous quantities of fat.

The purging may be accompanied by fever from the first, but often at the beginning the temperature is normal, and the attack has all the characters of an ordinary intestinal catarrh. Sooner or later, however, the temperature rises to 102°, or 103° or higher, and the general symptoms at once become severe. The child wastes quickly and grows very weak. His face is pale; his eyes get hollow; and the corners of his lips are encircled by a deep wrinkle like the mouth of a very old man. The tongue may be a little furred, but is usually clean and red and inclined to be dry. Sometimes vomiting is distressing; the pulse is then very rapid and feeble and the exhaustion great. The skin generally is dry and may be reddened about the buttocks and backs of the thighs by the irritation of the discharges from the bowels. The secretion of urine is scanty and, if the stools are profuse and watery or the vomiting urgent, no urine may be passed for twenty-four hours at a stretch, or even for several days. When this is the case I have known general œdema to occur. In a child of five and a half months old seen by me with Dr. W. H. Hatfield the secretion of urine was suppressed for four days, and the child, although not obviously swollen, pitted on pressure all over the body.

In a few days the depression of the child becomes extreme, and his fontanelle sinks deeply. His pulse is quick and very feeble; his breathing is hurried and shallow; his eyes are hollow; the lids are purple and close incompletely, and his face is livid. There is usually great thirst, and the tongue is dry and brown, or may be sprinkled with thrush. The feet and hands often feel cold although a thermometer placed in the rectum registers 105°, 106°, or even higher. I have known the mercury to rise as high as 109° in an infant who afterwards recovered; but such an extreme temperature is a very grave symptom, and if the heat mount rapidly from a moderate to a high level the patient often dies in a state of collapse.

When the inflammatory process affects especially the larger bowel (colitis) each stool is preceded by griping pains in the belly, and accompanied by much effort and straining. Often the bowel prolapses; and when the straining is urgent the protrusion may be difficult to replace. The stools appear gelatinous from excess of mucus, and may be streaked or stained with blood. On this account the complaint is often

spoken of as "dysentery" or "dysenteric diarrhœa." As a rule in this country such symptoms are easily controlled, but a bad case is met with from time to time in which the vomiting is distressing, the abdominal pain frequent and severe, and the tenesmus almost constant. The prolapsed mucous membrane then bulges from the body like a bright red glistening ball, and is shot out again at once when an effort is made to replace it. The stools are small and frequent, and consist of little else than mucus, either clear or tinged more or less deeply with blood. In this form the illness approaches the type commonly met with in tropical climates. The temperature may be high and the depression extreme, and the attack often ends fatally. If with high fever the vomiting is obstinate and the tenesmus urgent, we have reason to fear the worst.

In fatal cases of inflammatory diarrhœa death is almost always preceded by the signs of general collapse above described. In certain cases this condition may be due to the occurrence of parenchymatous nephritis; but it is exceptional after death to find obvious signs of inflammatory mischief in the kidneys. I believe, however, that an arrested secretion of urine and consequent imperfect depuration of the blood does often occur without leaving recognisable traces of organic change; for whenever there is a marked tendency to drowsiness and collapse the skin begins early to lose its elasticity. This change is best noticed on the abdomen where the skin on being pinched up lies in loose wrinkles as left by the finger and thumb. The urine in such cases is very scanty and difficult to obtain, but Kjellberg states that it is sometimes albuminous, and deposits epithelium and hyaline casts and small round cells. The child is very drowsy and stupid, but, if severely griped, may show signs of abdominal pain. Usually, however, he seems to suffer little if at all, but lingers on—perhaps for days—in a dull, apathetic state, careless of all that passes around him, until he ceases to breathe. In very young babies the drowsiness may deepen into coma, and the infant fall into a state which has been called "spurious hydrocephalus," from its likeness to the third stage of meningitis. Lethargy, at first partial only, soon becomes profound; the child lies straight on his back, with pinched, sharp features and a leaden-coloured face, breathing irregularly and sometimes heaving a sigh. His eyes seen between the half-closed lids are dull-looking; and the pupils are sluggish and often unequal. The fontanelle is deeply sunken, and if the infant be very young the bones of the skull can be felt to overlap. The nose, the feet, and the hands are cold, and sometimes the legs feel chilled as high as the hips. In the rectum the temperature is normal or below the level of health. In this state small greenish watery stools may still be passed without effort, but often when the coma begins the purging ceases. The most energetic stimulation will usually fail to rouse a child so profoundly depressed, and life is rarely prolonged beyond a very few days. Sometimes death is preceded by a convulsion. Symptoms such as these show a very sluggish state of the circulation in the brain, and in many cases, although by no means in all, they betoken a thrombosis of one or more of the cerebral sinuses.

After the age of infancy the complaint, although still a dangerous one, is yet of less gravity than before. The child has greater strength to face the weakening effects of the illness and runs less risk of grave complications. The nutrition of the patient, however, is at once arrested. He begins straightway to lose flesh and strength, his face becomes haggard, and his eyes are hollow. If vomiting be present also the features get pinched, the expression more distressed, and wasting goes on faster than before. There is usually thirst, and a craving for fluid may lead the patient to swallow beef-tea, milk, and other nourishing liquids; but he has no appetite for food. The child is often uneasy and restless, but there seems to be little actual pain unless the lower bowel be affected. The stools are watery and dark in colour, or may be yellow or green from excess of bile. Often they contain lumps of curd and masses of fat, and have a very offensive odour. The urine is high coloured and scanty and, according to Nothnagel, may contain excess of indican. If vomiting be urgent the renal secretion may be almost suppressed. In such cases the skin becomes markedly inelastic, especially over the belly; and the abdominal wall itself is often so sunken and hollow as to resemble that of a case of tubercular meningitis. The temperature is either elevated throughout the attack, as in the case of younger children, or falls after the first few days to a level below that of health. If the case end badly death is generally due to exhaustion; therefore a patient who is already weak when the attack begins has necessarily a less chance of recovery. For this reason inflammatory diarrhœa, occurring as a complication in the course of another illness, must always give rise to much anxiety.

The *duration* of acute entero-colitis is usually short. A fatal attack rarely lasts longer than a week, and often comes to an end on the third or fourth day. The immediate danger is, of course, in proportion to the violence of the symptoms; but it does not follow that a case which begins mildly must always end in recovery. The illness may drag on for days or even weeks, varying in intensity, but gradually bringing the child lower and lower until he sinks from exhaustion. These, it must be owned, are usually the cases where the patient is regularly washed or exposed in other ways to chill. On the other hand, we sometimes see the attack begin violently enough with high fever and frequent watery stools; but after a day or two the temperature falls, the purging abates, and all danger is at an end. These abortive attacks are sometimes described as a special variety of the complaint. Of patients who survive some mend quickly; others are left pale, feeble, and subject to catarrhs; others, again, suffer for months, and even die at last of a chronic intestinal derangement which is often complicated with intestinal ulceration, and sometimes dependent upon it.

Complications.—There are certain intercurrent lesions which may be met with, especially in the more protracted cases, and add to the danger of the illness. Simple and parasitic stomatitis are not rare, and increase the difficulty of feeding the patient. Pulmonary catarrh often comes on; and as the child becomes exhausted, collapse of the lung is apt to occur,

and helps to shorten his life. Catarrhal pneumonia has already been referred to. According to Lesage it is due to direct infection of the lungs by breathing air tainted with the virulent bacillus coli. As it greatly increases the weakness of the patient and hastens the end the pulmonary inflammation at the time of death is usually still in an early stage. On this account it often passes unheeded, especially as the great weakness of the patient forbids complete examination of the chest.

A few twitching movements may be noticed before the end, but are not common. Sometimes violent convulsive seizures occur early in the illness, and are followed by a state of stupor from which the child can never afterwards be roused. As far as my experience goes this only happens in cases where the patient is living in dangerously insanitary conditions. Some years ago I saw with Dr. Ford Anderson a little girl of fifteen months old, who had been seized early on June 9th with looseness of the bowels. In the course of the afternoon the child had a fit, and in the evening the convulsions returned and the patient lay twitching, on the right side only, for seven hours. After the nervous symptoms had subsided the child remained in a stupor. The temperature was uniformly high and on the second day was 105° in the rectum. On 12th June, the fourth day of the illness, the rectal temperature was 103·6°. The bowels were then acting seven or eight times a day, the stools being loose and green, with some curd-like lumps and a little mucus. The child lay in an unconscious state with eyelids partially closed. The cheeks were a little flushed and the complexion was of a smoky tint. The pupils were equal, of normal size, and acted well with light. The abdomen was a little flaccid, but not retracted or doughy to the touch. The state of the house in which the child was living was known to be insanitary, and the mother had suffered from diarrhœa several days previously. The little girl died on the fifth day without any return of consciousness. Dr. Ford Anderson examined the body after death, but found only swelling and redness of Peyer's patches and universal fluidity of the blood. The brain was healthy.

An acute otitis media sometimes arises in the course of an intestinal affection and may set up serious cerebral symptoms. Therefore in all cases where such symptoms appear the ears should be examined with a speculum and reflected light. I have known delirium, convulsions, rigidity of the muscles of the neck, and a condition approaching coma to cease quickly when a perforation of the tympanic membrane has allowed pent-up pus to escape from the ear cavity.

Diagnosis.—Before pronouncing a case to be one of inflammatory diarrhœa we must be careful to exclude other feverish complaints which sometimes start with digestive troubles. Pneumonia and some of the eruptive fevers may begin in this way. The skin then must be searched for signs of rash, and as we proceed with our examination the early symptoms of measles, scarlet fever, and the like must be kept in mind. An acute entero-colitis is not often confused with typhoid fever during the first few years of life, for the latter complaint is rare in early childhood,

and when it occurs is accompanied more often by confined bowels than by diarrhœa. In the older children, however, there may be some uncertainty upon the point; but in entero-colitis the abrupt onset and shorter course of the illness, the severity of the purging from the first, the pinched expression of the patient, and, as time goes on, the absence of rash or of splenic enlargement, should be sufficient to exclude the specific fever. It may be remarked that a "pea-soup" look of the motions for a day or two is in itself no proof that the complaint is typhoid. Stools of this kind are not uncommon in the diarrhœas of children, and we must not be deceived by them, especially as a trained nurse may cause some trouble by confidently pronouncing the case on this symptom alone to be one of enteric fever.

The seat of the inflammation in the bowels may often be inferred from the appearance of the stools. Nothnagel has shown that mucus is always present in the evacuations, and, when invisible to the eye, can be detected by the microscope. The higher in the intestinal tract the inflammation is seated the more intimate is the admixture of the mucus with the fæcal contents of the bowel. In a catarrh limited to the upper part of the bowel no mucus is visible to the naked eye, but it can be detected by the microscope. In such a case the colon may be taken to be healthy. If under the microscope the mucus is seen to be tinted with bile we infer that the jejunum and ileum are also involved; the presence of bile indicates also that there is increased peristaltic action of the muscular coat forcing the contents along, for if the bile linger in the bowels it becomes changed in appearance and ceases to respond to Gmelin's test. If mucus be plainly visible to the naked eye the lower bowel is always affected. Pulpy stools containing dabs and drops of free mucus point to the colon; and pure mucus in large quantity to the sigmoid flexure. Scybala imbedded in mucus show catarrh of the rectum, in which case painful tenesmus is almost invariable.

It is scarcely possible in a case of entero-colitis to be quite sure as to the exact pathological condition. Ulceration is probably present in the more protracted cases only, and even in these must not be too hastily assumed. Blood-stained stools, as Dr. Donkin has well pointed out, cannot be held by themselves to imply actual breach of surface, since oozing of blood from a merely congested mucous membrane is far from uncommon. Still, attacks of colicky pain preceding the stool, with some increase of tension of the abdominal wall, are symptoms which should excite inquiry. If, in addition, there be moderate tenderness on deep pressure along the course of the colon, and especially if small dark clots can be detected in the stools, the presence of ulceration of the larger bowel may be more than suspected.

Prognosis.—An attack of inflammatory diarrhœa is always a serious thing, and if the complaint occur in an infant, especially in one whose digestive organs are continually overtaxed and teased by ill-chosen food, disastrous consequences may be expected. The prospect, therefore, is less bright in infancy than in later life, and is gloomy in proportion

to the degree to which the nutrition of the patient is impaired. The temperature (taken in the rectum) is a good test of the gravity of the child's condition. If it remain at or above 105°, or shoot up suddenly from a low to a high level, the danger is great; on the other hand, a fall in the thermometer is a hopeful sign; and if the bodily heat become normal (in the rectum) we may look for recovery, although no immediate improvement be noted in the number or character of the stools. In addition to a high temperature other signs of serious import are:—persistence of the vomiting, heaviness and tendency to collapse, noisy breathing, convulsions, loss of elasticity in the skin. The last symptom is one which it is important to watch closely, for little improvement can be expected until the elasticity of the skin is restored.

C. Choleraic or septic diarrhœa (Summer diarrhœa; Cholera infantum).—This form of the illness is especially a complaint of hot weather and crowded cities. Although not unknown in later childhood it is in infancy, during the cutting of the milk teeth, that the disorder is most prone to occur; and from its rapid course, its grave symptoms, and its too commonly fatal ending, is one of the most justly dreaded of the illnesses of early life. It is a noteworthy feature of the complaint that the patients are so often sturdy, well-nourished babies; for strong children are not less likely to suffer from it than weakly ones, and when seized may die almost as quickly. I think boys are more subject to it than girls.

Symptoms.—The complaint comes on quite suddenly. The child may have seemed quite well up to the time of the attack, or may have suffered for a few days from a slight ordinary looseness of the bowels. All at once, however, he is found to look pale, pinched and ill. He vomits, throwing up first the food he has taken; afterwards a thin watery mucus coloured more or less deeply with bile. His motions, in the beginning thin, fæculent, and very offensive, soon turn to mere serous fluid, which loses for the most part its repellent odour, and leaves only a yellowish stain with no trace of fæcal matter upon the diaper. The action of the bowels is not attended by any griping or tenesmus, but the frequent profuse stools so drain the tissues of fluid that the child falls away with startling suddenness. After an absence of only a few hours we find him so changed that we hardly know him again. His eyes are sunken, his cheeks are hollow, and his nose is pinched and thin. His body, too, has shrunk up and his flesh feels soft and doughy. The skin is inelastic, especially over the abdomen; often it lies there in loose folds, and the abdominal wall may be as deeply hollowed as in the most pronounced case of tubercular meningitis. Thirst is of course extreme. If the child can talk he asks continually for drink. If he be an infant, he sucks his lips and lies whining fretfully with his eyes fixed upon any cup or vessel holding fluid. But his thirst is little relieved by what he takes; indeed, anything swallowed is usually returned in a few seconds. His lips are dry, and the wrinkle which curves on each side round the angle of the mouth makes them seem to project. The

tongue, if clean and moist at first, soon becomes dry and brown. The urine is scanty and may be quite suppressed. The pulse is rapid and weak. The temperature is high. Even when the skin is cool to the touch and the feet are quite cold a thermometer held in the rectum will often register a heat of 104° or 105°. On this account the temperature of the axilla or groin must not be taken as a guide to the internal heat of the body. At first the patient is very restless and cannot sleep; he throws about his arms and whimpers feebly as long as he has strength to do so; later, he lies in a drowsy state with dull eyes and half-closed lids taking no notice of any one. His fontanelle is deeply sunken, and in a young infant the bones of the skull can be felt to overlap.

Vomiting now usually ceases, but small watery motions continue to be passed at intervals. Very soon the patient sinks into a state of collapse. He lies quietly on his back in his cot, his eyes showing white between his half-closed purple lids. His face looks thin and pinched, and is wrinkled like the face of an old man. The complexion is earthy or lead-coloured, and his extremities, and even his nose, feel cold to the touch although the internal temperature is still high. In this state all reflex phenomena are abolished. The patient is insensible to all efforts to feed him, and seems unable to swallow; the sensitiveness of the conjunctiva is lost; the faint breathing alone shows him to be alive, and this soon comes to an end. A few feeble convulsive movements may precede death, and the temperature may rise to 106° or 107°.

The course of the complaint is very short; it may last from a few hours to a few days. Almost always before the end of a week the child has either ceased to live or has turned the corner and begun to mend. If a favourable change occur we first notice a fall in the temperature; then vomiting ceases and the patient begins to retain fluids; he is less thirsty, and his motions become coloured again with fæcal matter or bile. The looseness rarely ceases suddenly, but thin feculent stools continue to be passed, although less often and in smaller quantity. Sometimes the improvement in the stools is noticed before the vomiting has stopped, but the usual course is that described.

Diagnosis.—Choleraic diarrhœa is easily recognised. The intense thirst and obstinate vomiting, the sudden falling away as the tissues shrink up, the copious serous stools without a trace of fæcal matter, and the early collapse form a very characteristic group of symptoms.

Prognosis.—The prospects of a patient stricken with this complaint are never otherwise than gloomy. In young babies especially the rate of mortality is very high. The danger is in proportion to the height of the temperature, the urgency of the vomiting, the frequency of the stools, and the degree to which the elasticity of the skin is impaired. The last symptom is one which should always be carefully noted; even towards the beginning of the complaint, when the other symptoms, perhaps, do not denote any very pressing danger, if the skin is found to be inelastic we must look to the issue of the illness with grave misgivings. If the child become collapsed, the prospect is even more gloomy than in cases

of ordinary inflammatory diarrhœa. In the latter complaint the patient may often be roused by energetic treatment; but in choleraic diarrhœa a favourable change is very difficult to establish, and if the patient be a very young child or infant recovery can hardly be anticipated. On the other hand, any abatement of the symptoms is to be welcomed as a valuable sign of improvement. Early cessation of the vomiting, a return of fæcal matter to the stools, or, in particular, a fall in the internal temperature of the body, furnish solid ground for hope that the patient may eventually recover. But in infancy a favourable issue to the illness must always come to us rather as a surprise, especially as a good state of nutrition and previous sound health seem to influence the prognosis but little. Collapse may show itself earlier in a weakly infant and later in a strong one, but in each case the end, when it comes, is apt to be the same. Fortunately in older children recovery is more common; and the outlook may be taken as less and less sombre in exact proportion to the advance in years.

Prevention of diarrhœa.—In the large majority of cases the diarrhœas of children may be looked upon as a consequence of want of knowledge or want of care. At least they may generally be prevented by attention to the feeding and management of the child, the cleanliness of his nursery, and the healthiness of his surroundings. This is especially the case in hot weather, when the worst kinds of bowel complaint are likely to be met with. To protect a child from diarrhœa we should put him into the best conditions for health. If he live in a crowded city he should be removed in the summer to an airy, well-drained house in some country place. Here he can spend his days out of doors, drink new milk, and sleep in a pure atmosphere. If he cannot be sent out of town we must do our best to keep him from bad air and stale or tainted food. The child should pass as much of his time as possible in the fresh air, spending a large part of the day in the parks, and sleeping at night in a well-ventilated room. The sanitary arrangements of the house must be carefully looked to: water-closets must be kept clean and regularly disinfected; attention must be paid to the housemaid's sink, which should not be used for the disposal of bedroom slops; soiled linen must never be allowed to remain in the nurseries; and care must be taken that the child's living and sleeping rooms are not near to an open drain-pipe, however thoroughly this may be trapped or cut off from the sewer. Many an attack of diarrhœa has been set up by a water-closet or housemaid's sink just outside the nursery door.

The child should be carefully guarded from chills. The washing of his body must be a rapid process, and he cannot be allowed to prolong his bath unduly or play in the water. At night he should be lightly covered, and if he toss about in his sleep means must be taken to prevent his throwing his coverings aside and lying naked in his bed. The day dress is not to be neglected. Many young children suffer greatly from too scanty clothing, and especially from bare legs. Even in the summer the rapid changes of temperature inseparable from our climate are full of risk to an ill-protected child. If there be any suspected

delicacy of constitution, or known susceptibility to colds, the legs should be covered with long woollen stockings, and the hips and thighs with woollen drawers.

In the matter of feeding we must see that all jugs, cups, feeding-bottles, and the like, are kept clean, and that no food but such as is wholesome and in good condition is provided. It is best to boil or sterilise the milk without loss of time and put it aside in a refrigerator or some cool place. It must not be allowed to stand in a living-room. All meals must be freshly prepared, and it is unsafe to keep milk through the night for the early meal in the morning. Before the fresh milk is brought to the house it is wise to employ condensed milk or one of the desiccated milk foods.

In sultry weather the child, if thirsty, is not to be deprived of water; but this should be boiled and filtered. An infant may suck water from his feeding-bottle, and older children may be allowed to drink freely between their meals. Milk must be looked upon strictly as a food and not as fluid to be swallowed whenever the child is thirsty. The summer fruits should not be given to infants, but if ripe and sound and freshly gathered may be included in the dietary of older children. Unripe, stale, or tainted fruits must not be taken on any account. Meals should be fixed at regular intervals, and should not be too large. It is unwise to overload a child's stomach at any time, but in hot weather excess of saccharine or starchy food is especially liable to promote acidity and set up diarrhœa.

Treatment.—In the treatment of diarrhœa in the child we have to put a stop to the drain from the bowels, to lessen or remove the ill consequences arising from it, and to restore the normal processes of nutrition. If the complaint be a mere catarrhal derangement of little moment its management is an easy matter; but if the whole system have been contaminated by the absorption from the bowel of the products of poisonous decompositions, our utmost ingenuity and resource may be taxed to guide the complaint to a favourable issue.

In the case of a mild catarrh of the intestine our first care must be to keep the child in an equable temperature and to avoid every source of chill. While the catarrh lasts the natural sensitiveness of the body to changes of temperature is heightened, so that the derangement once started may be prolonged from day to day by an exposure which in the beginning would have been powerless to set it up. For this reason the belly must be covered with a flannel binder, and while the purging lasts a general washing of the body must be forbidden, although for the sake of cleanliness rapid local sponging after a stool may be allowed.

If we have to do with an infant and find him crying and drawing up his legs with abdominal pain, while the motions consist of undigested food and mucus, an aperient dose of castor oil or rhubarb and soda should be ordered at once to clear away the irritating matters. The laxative may be repeated if at any time undigested food be found in the stools, or the belly become hard and distended. After the action of the aperient the

infant may take a few doses of the time-honoured chalk and catechu mixture. This is especially indicated when the stools are frothy and sour-smelling. If preferred, a few drops of tincture of rhubarb may be given, with half a drop of laudanum and a drop of sal volatile in an aromatic water. For diet the child, if not at the breast, should be fed with milk and freshly made barley water in equal proportions, alkalinised with fifteen or twenty drops of the saccharated solution of lime.

This treatment will quickly put an end to an ordinary simple diarrhœa; but if the child continue to be bathed as usual, or be in any other way exposed to chill, the purging may resist the remedies, or return after apparent arrest. If this happen, three or four grains of rhubarb with half the quantity of aromatic chalk must be given at bedtime, and the dose may be repeated on the following night if the stools remain frothy and sour-smelling. In the daytime a twelve months' old child may take oxide of zinc (gr. j.), or carbonate of bismuth (gr. v.) and aromatic chalk (gr. j.) three times a day; or the rhubarb and sal volatile mixture as directed above. If, later, the stools become thin and watery, a little tinct. opii (♏ss.-j.) can be added to the dose. Astringents are useful at this stage, and the opiate is of especial service. The treatment must be continued as described under the head of inflammatory diarrhœa.

In cases where the catarrh occupies the lower bowel, and slimy stools are passed with straining efforts, small doses of castor oil and opium are indicated. Two drops of the oil with one of ipecacuanha wine and half a drop of laudanum may be given to a twelve months' infant three times a day. This form of looseness, and indeed any kind of simple diarrhœa, may be kept up by irritation low down in the rectum. Therefore in cases which do not yield readily to treatment it is advisable to order a small injection (two drachms) of thin warm starch to be thrown into the bowel immediately after each motion.

Green stools, if acid or loaded with bile, require alkaline remedies. The green stool which is neutral, or only slightly acid, and gives no biliary reaction, can often be quickly changed for the better by a teaspoonful of a 2 per cent solution of lactic acid (as recommended by Hayem) given every four hours. In all cases soiled diapers should be removed from the room, and indeed from the neighbourhood of the nursery, without loss of time; if a green tint is noticed in the stools it is well to steep the napkins in a solution of corrosive sublimate (1 per 1000).

The looseness of the bowels which is common in teething infants must not be allowed to continue. Purging during dentition is in no way a beneficial process, but tends still further to reduce a child who is probably already depressed by pain and want of sleep. A weakly infant whose bowels are irritable and often relaxed may lose strength to an alarming degree after but a few days of the derangement.

After the age of infancy simple diarrhœa must be treated much in the same way as described above. While the derangement lasts the

child should be confined to the house and made to discontinue his ordinary washing-bath. In the matter of diet he must avoid acid-making things, such as fruit and sweets. For medicine he should take an aperient dose of castor oil or rhubarb and magnesia; and when irritating matters have been cleared away he may begin an aromatic and antacid draught of sal volatile and chloric ether with a drop or two of laudanum in cinnamon or peppermint water. This draught taken three times a day will soon put a stop to the disorder.

Lienteric diarrhœa is very amenable to treatment. For a child of five years old, one drop of Fowler's solution of arsenic and the same quantity of tincture of nux vomica may be given before meals three times a day, in a draught with dilute nitro-hydrochloric acid and infusion of calumba. At the same time the abdomen must be protected with a substantial flannel bandage; the feet must be kept warm; and every source of chill must be carefully guarded against.

In a case of *inflammatory diarrhœa* the patient should be put to bed at once. Even an infant or young child is better in his cot than resting on the lap of the nurse, heated by contact with her body and breathing her used air. The abdomen must be covered with a flannel binder, and the daily washing-bath must be put a stop to.

In the matter of *diet* it is of great importance for the time to give up the use of foods which are capable of decomposing to form poisonous ptomaines. Unless in the case of a breast-fed infant milk is to be forbidden; and even infants reared entirely on the breast will sometimes cease for the time to digest their mothers' milk. Bottle-feeding with milky foods is out of the question, however carefully the milk may be sterilised. Instead, the child should take freshly-made whey diluted with an equal proportion of barley-water, or weak veal or chicken broth, mixed with an equal quantity of fresh and fresh-barley water. These foods may be given cold in alternate meals in small quantities at short intervals. If the infant be very weakly or show signs of exhaustion, white wine whey should be given in doses of one tablespoonful as often as may seem desirable. If the weakness be great, the child may take no other food until his strength improves. As he begins to mend, Mellin's Food may be added to the meals, but milk is not to be resumed until convalescence is well advanced, and then only with great caution.

An older child can be fed with small meals of whey, barley-water, broth, or Denayer's meat peptones diluted with barley-water. If the strength show signs of failing, the brandy-and-egg mixture (B.P.) can be given. Still, in this matter of feeding we must remember that the weaker the child the more feeble his digestive power, and that food must be given in small quantities only. The strong jellies and extracts of meat must be well diluted with water, and every care must be taken not to overtax the feeble organs. As a rule, however, the patient is more eager for drink than for food, and at any age may be allowed to slake his thirst with cold water (boiled and filtered), in moderate quantity, as often as he wishes for it. Infants must be carefully watched

for signs of thirst, and in any case notable shrinking of the tissues is a sign that water is urgently needed.

The air of the room must be kept fresh. In suitable weather the window must be opened, taking care that the child's cot is out of the direct line of draught. But few persons should be allowed in the room, and all soiled linen must be taken away at once.

In all cases of inflammatory diarrhœa the bodily heat must be carefully noted and reduced if it rise too high; it should be kept below 103°. In taking steps to lower the temperature we must remember the tendency in this complaint to sudden collapse, and have stimulants at hand to correct any sudden failure of the heart. The temperature may be brought down by sponging with tepid water, by placing the patient in a bath of 90° F., or by enemas of cooled water. Of these methods I prefer the last if the child can bear it. The shock is in exact proportion to the coldness of the water, and therefore it is best to begin with the use of water cooled down to a temperature of 80° F., three or four ounces of which may be thrown into the bowel. If this be well borne the temperature of the enema may be slightly reduced on the next occasion. Ice-cold water has been recommended; but injections of this temperature must be used with great caution in a complaint where the tendency to sudden collapse is so decided. Too rapid a reduction of the bodily heat may be attended with alarming symptoms of heart failure, and I have known a child's life to be brought into imminent danger by this means. Even at a temperature of 80° the injection has a markedly lowering effect upon some children, and we must be vigilant, therefore, to note any sign of depression. It is wise to administer a dose of brandy both before and after the operation, and to place a hot bottle wrapped in flannel against the child's feet when he is returned to his cot. If necessary, a hypodermic injection of four or five drops of ether may be given.

If the elasticity of the skin be impaired we must take steps at once to restore its healthy action. To gain this end I know of no plan more useful than the wet pack. The child is wrapped as high as the armpits in a towel wrung out of cold water (made stimulating with a sixth part of eau-de-Cologne or brandy) and is then closely covered from the neck downwards with dry blankets well tucked in. The patient may be kept thus swaddled for six, eight, or ten hours, or longer; but every three hours he must be taken out, rubbed dry, and quickly repacked as before. A dose of spirits of nitrous ether (♏ v.-x.) given in a little water every two hours during the process is a great help in this treatment. I look upon the state of the skin as due to faulty action of the kidneys, and have found nitrous ether quite without an equal as a quick and certain diuretic for young children. Besides helping to restore the normal suppleness of the skin the wet pack also tends to lessen the fever. In certain cases, however, the contrary effect is noted, and the bodily heat rises instead of falling. If this happen the wet pack must be abandoned as hurtful, and we must be contented to trust to the nitrous ether alone.

The use of *internal remedies* must go hand in hand with the other

methods of treatment. As a first step, if the child be seen early, a dose of castor oil is always useful to clear away irritating matters from the bowel; but when this has been done we seldom find benefit from the ordinary astringents so long as the bodily temperature remains high. Although no definite rules for treatment have been arrived at as a result of bacteriological inquiry, some form of antisepsis of the bowel, if this can be attained, seems to offer the best prospect of success. With the exception of calomel, however, antiseptic remedies, it must be confessed, have not as yet given satisfactory results. I have made use of salol, β-naphthol, naphthalin, resorcin, iodine, carbolic acid, and the salicylates, but can boast of but little advantage from their employment. Given alone they have seemed to me to be almost inert. I have thought them of service only when combined with small doses of calomel or gray powder. After a fruitless trial of new remedies I have had again and again to fall back upon the old-fashioned combination of calomel and Dover's powder given in frequent small doses, and believe it to be the best form in which an intestinal antiseptic can be prescribed. In addition to the germicide action of the mercurial, the opium is useful in regulating the extravagant peristalsis, and the ipecacuanha has a very beneficial action upon the mucous membrane of the bowel. I give a quarter of a grain of each every three hours to a one-year-old infant, and the improvement is often so striking and immediate that I cannot but attribute it to the action of the remedy. I have tried calomel in combination with most of the antiseptics already enumerated, and the perchloride of mercury with resorcin, but with less obvious benefit. Ipecacuanha has long been esteemed for its influence in improving disordered states of the intestinal mucous membrane, and is, indeed, as useful in derangements of the bowels as in catarrhs of the lungs. If the colon be the part affected, or if there be excessive vomiting, ipecacuanha should on no account be left out of the prescription.

When the presence of mucus in the motions shows the lower bowel to be affected, medication by the rectum becomes useful. Irrigation of the bowel by warm saline solutions is of great service in these cases. In the East London Hospital for Children, with the help of the resident physician, Dr. R. P. Cockburn, I made large use of this method of treatment during the summer of 1896, and found that if injected slowly and gently, so as to throw in a large quantity of fluid, the irritability of the bowel was appreciably diminished, the stools were reduced in number and improved in quality, and, moreover, a certain absorption of fluid took place from the bowel, giving a perceptible increase of fulness to the frame. The strength of the solution used was one teaspoonful of common salt to the pint of warm water. The irritability of the lower bowel may also be lessened by the injection of small quantities of laudanum (♏ij.-iij.) or cocaine (gr. $\frac{1}{6}$) in two teaspoonfuls of thin warm starch after each motion; or by the use of suppositories containing the same quantity of sedative. When the rectum is acutely inflamed and prolapses like a bright red glistening ball which cannot be replaced, the protruded

part must be bathed after each stool with warm water, and then covered with a thick poultice of boiled starch. Twice a day the laudanum and starch injection may be used with the addition of five grains of powdered ipecacuanha before applying the poultice. At the same time small doses of castor oil (♏ij.-iij.) should be given with half a drop of laudanum and one drop of ipecacuanha wine in a little aromatic water every four hours. Vinum ipecacuanhæ in the same dose, but given every hour, will usually arrest the vomiting, which is often a distressing feature in these cases; or the same object may be gained by one-fifth of a grain of cocaine given several times in the day.

If prolapsus ani continue after the inflammation has been reduced, or come on later from relaxation of the sphincter and irritability of the mucous membrane of the rectum, the protruded bowel must be sponged and returned after each motion, and a small cocaine injection can be given as recommended above. If the mucous membrane become flabby as a result of repeated catarrhs of the bowel, an enema of infusion of rhatany or of nitrate of silver (gr. ij. in 4 ounces of water) used every night will usually put an end to the trouble, after a few repetitions.

When the fever has subsided the diarrhœa often becomes amenable to ordinary astringent remedies. Of these I prefer the extracts of hæmatoxylon (gr. j.-iij.) and rhatany (gr. j.-iij.) and the tincture of catechu (♏v.-x.) to gallic acid or lead. Bismuth and aromatic chalk also form very useful remedies, and opium should always be added if there is any undue force in the expulsion of the stool. To be successful the dose of bismuth should be considerable, and as the action of the drug is purely local, large quantities may be given to quite young children without fear of ill consequences. I prefer the subnitrate to the carbonate, and give it in doses of not less than 10 grains every two, three, or four hours, combined with a grain of aromatic chalk and a small dose of resorcin or other antiseptic. If, as may happen, all astringents seem to irritate, the old prescription of dilute nitric acid and opium is very useful. For a child twelve months old two drops of dilute nitric acid may be given with half a drop of laudanum in water sweetened with glycerine three times a day. The same cases are often benefited by the sal volatile and laudanum mixture recommended for simple diarrhœa.

When signs of prostration are noticed they must be attacked without loss of time. The child should be put into a hot mustard bath at once and held there for five minutes, or less if the blueness disappear earlier from his lips. Afterwards he is to be wrapped in flannel, and returned to his cot with hot bottles to his feet and sides. Every hour or half-hour a teaspoonful of the brandy-and-egg mixture, or white wine whey (according to the age), should be given; and, if exhaustion be great, a weak mustard and linseed-meal poultice can be applied for several hours to the chest and epigastrium. The mustard bath, although it will raise a lowered temperature, will not reduce the bodily heat when this is high; therefore the fever should be brought down by injections of cooled water as already recommended.

In the summer of 1896, with the assistance of Dr. Cockburn, at the East London Hospital for Children, I made trial of various cardiac stimulants in the hope of finding some effectual remedy to rouse the patient from his collapsed state. Of these camphor, which has been so highly recommended, seemed of little value unless given hypodermically with ether. The inhalation of oxygen was quite useless. The drug which produced the most immediate and striking effect was strychnia. The $\frac{1}{400}$ of a grain—that is, a quarter of a minim of the Pharmacopœia solution—introduced under the skin seemed to give some life to the infant; but the effect only lasted about three-quarters of an hour, after which time the dose had to be repeated. We found that if given at shorter intervals a certain rigidity of muscles was produced by the remedy. In cases of pronounced collapse I believe this method of treatment to be a useful addition to our resources. When the patient is merely exhausted without being collapsed, a few drops of ether, given hypodermically, have a strongly stimulating effect. When the child is prostrate he must be kept in a horizontal position, and the nurse should be forbidden even to raise his head from the pillow.

In cases of recovery the mucous membrane is left in a relaxed state, and requires bracing up by tonic remedies. A few drops of the liquor ferri pernitratis are very useful at this time, given well diluted and in combination with tincture of nux vomica. During convalescence, and indeed for months afterwards, the susceptibility of the patient to fresh chills must be kept in mind, and he should be dressed warmly in woollen underclothing, and wear a flannel band to his belly.

The treatment of *choleraic diarrhœa* is greatly hampered by the obstinate vomiting, which not only forbids the stay of medicines on the stomach but baulks all our efforts to supply much-needed nourishment. So long as this excessive gastric irritability continues it is best to hold over all attempts to feed the patient, and to content ourselves with giving iced water, in small quantities, as often as the child is willing to swallow it. The liquid is usually returned at once, but the thirst is so extreme that it is cruel not to do our best to satisfy it.

In all these cases the skin is excessively inelastic, and no time should be lost in packing the child in the wet towel as already recommended for inflammatory diarrhœa. If the vomiting be urgent, a poultice containing one part of mustard to five parts of linseed meal should be applied to the epigastrium before using the wet pack. In addition, one-sixth of a grain of calomel may be placed on the tongue every half-hour. Sometimes the vomiting stops after a few repetitions of this dose, especially if the stomach has been previously washed out, as recommended by Epstein. This operation is an easy one, and consists in introducing a French elastic catheter (No. 7), through the nose into the stomach, as the child sits on the nurse's lap. Four ounces of boiled water at a temperature of 95° are then passed through the tube and allowed to flow out by bending the child forwards. The process is repeated several times until the fluid returns quite clear from the stomach. Dr. A. Seibert, who has used this

method of treatment in a good number of cases, speaks highly of its efficacy, and I have myself been pleased with the results when used in addition to the other means I have described. Unfortunately the operation requires so much movement, that it can only be employed at the very beginning of the illness, before the child becomes exhausted.

Instead of calomel other salts of mercury may be made use of, such as the perchloride or biniodide. Dr. A. P. Luff speaks favourably of the latter remedy in doses of gr. $\frac{1}{50}$, with 1 grain of chloral hydrate given every three hours to a child between the ages of six and twelve months. If other means fail, resorcin may be tried in doses of 2 or 3 grains every three hours. The stomach is sometimes quieted by a few doses of this remedy, but the drug has little or no influence upon the diarrhœa.

We must take advantage of any pause in the vomiting to supply food. This must be given at first in small quantities and in the simplest form. I prefer white wine whey, which combines food and stimulant, given from time to time in a teaspoon; but plain, freshly made whey, veal broth and fresh barley-water, koumiss, or Denayer's sterilised meat peptones well diluted with water may be used, with the addition of five or more drops of pale brandy. The foods must be used cold, and if the quantities first allowed be kept down, may soon be given more liberally. Many children after some hours will take half a bottleful of white wine whey without harm at one meal; but any return of the vomiting should make us fall back at once upon the smaller quantities first recommended. Whatever kind of nourishment be chosen, it must be looked upon as food to be given at stated intervals, and not as drink to slake the thirst. For the latter purpose cold water is to be used, as already advised. If the patient become collapsed and the rectal temperature be high, the fever must be reduced by the means recommended for inflammatory diarrhœa. If cold enemas be employed, the temperature of the water at first should not be lower than 80°, and stimulants must be given freely both before and after the use of the syringe. Hot bottles may be required in the cot if the fall of temperature after the injection is considerable, and the hot mustard bath or even hypodermic injections of ether may be necessary. In addition to their influence in reducing temperature, the rectal injections are of service in supplying a certain amount of fluid to the tissues. I have tried hypodermic injections of the saline solution with the same object, and have found that 2 ounces injected into the loose tissue of the back were absorbed slowly, and seemed to give a little relief; still, so long as the exhausting discharges from the bowels continue, the small amount of fluid which can be introduced by the skin can do little to satisfy the wants of the system.

When the vomiting has been put a stop to, measures can be taken to arrest the purging, and any of the remedies recommended for inflammatory diarrhœa may be made use of. I have had good results from the subnitrate of bismuth, but the quantity given must not be less than 10 grains every three or four hours to a child of twelve months. I usually

combine each dose with 2 grains of salicylate of soda and half a drop or so of laudanum.

If the patient be seen early, a hypodermic injection of morphia will often cause an immediate improvement in the more alarming symptoms, and moderate the vomiting and purging at once, or even completely arrest it. This, in children of four or five years old and upwards whose strength enables them to withstand the enfeebling influence of the complaint, may be sufficient to determine a favourable issue. Infants and the younger children, however, have not this reserve power and are more easily depressed. In them, therefore, although the irritability of the stomach and bowels may be held in check, the course of the illness is often not materially improved, and the patient sinks into a state of collapse and dies just as if the vomiting and diarrhœa had not been interfered with. The chances of recovery are much greater if the remedy can be adopted quite early in the illness. If it be delayed until exhaustion sets in, it may seem merely to add to the weakness of the patient. One thirty-fifth to one-fortieth of a grain may be used for an infant twelve months old combined with five drops of ether; and the dose may be repeated in two or three hours if a sufficient effect have not been produced. It is better to administer the narcotic in small doses at short intervals than to begin with a considerable dose at first, for in the beginning it is impossible to tell how susceptible a young child may be to the action of the remedy. If the child become collapsed, stimulating hypodermic injections of strychnia must be used as directed for inflammatory diarrhœa.

CHRONIC DIARRHŒA.—Chronic diarrhœa is a very dangerous and insidious form of disease which yields reluctantly to treatment and is often fatal to infants. It may be the sequel of an acute attack, or come on by slow degrees in a state of health. In the younger subjects the complaint is usually at first a mere catarrhal derangement. It may be so likewise in older children, but in the latter it is often the result of tuberculous ulceration of the bowel.

Etiology.—In infants acute and chronic diarrhœas are set up by the same causes; and acute diarrhœas often become chronic if the agency which first started the derangement continue to operate. Most cases of persistent diarrhœa are the consequence of chilling of the surface. The leisurely way in which the washing of an infant is so often carried out, without any reference to the state of his health, is no doubt answerable for many illnesses; and an intestinal catarrh thus induced may be kept up from day to day and week to week by continually recurring impressions of cold. A child who lives in this state of catarrh almost always has a languid circulation; and his cold feet and legs—exposed as they are too often to every change of temperature—make him keenly susceptible to chill.

In the same way an intestinal catarrh set up by the cor
of the mucous membrane by fermenting food may be pro

indefinitely by a persistence in the same diet. This is well seen in cases where feeding with milk and starch is not given up. There are few cases of chronic diarrhœa in the child which do anything but badly upon a milky or farinaceous diet. Such foods supply material for the fermentative action of bacteria. The microbes attack the milk-sugar and carbohydrates generally, and give rise, as Escherich has pointed out, to the formation of acetic and lactic acids, carbonic acid and hydrogen.

Even in older children chronic diarrhœa is frequently the consequence of cold feet or careless exposure in the bath. It is sometimes also a sequel of the infectious fevers and whooping-cough; but in these cases, too, I believe the exciting cause to be chill acting upon a system rendered more than commonly susceptible by the late illness. In strumous and tubercular children a chronic diarrhœa may be kept up by tuberculous ulceration of the bowels.

Symptoms.—When the chronic complaint follows an attack of acute diarrhœa the child, although the more urgent symptoms have subsided, remains weak and pale; his temperature is normal or even lower than in health; his appetite is poor; and his bowels discharge several times a day a thin, dark, offensive fluid or sour-smelling pasty-matter with mucus.

It is more common, however, for the disorder to begin gradually. The patient—usually a child of fifteen or eighteen months—is noticed to be looking pale and dull. His flesh is flabby and his feet are cold. His spirits, however, keep good, his temperature is normal, and he sleeps well at night. Soon he is noticed to be obviously thinner, and his strength begins to fail so that his love of movement leaves him; he is never happy out of his nurse's arms, and, if previously able to walk, may refuse to stand or even to rest his weight upon his feet when held to the ground. This change in his habit excites a good deal of comment and some anxiety, but the child's appetite continues good and his bowels —the nurse will declare—are "nicely open." In all such cases the stools should be inspected, and it will be found that some two, three, or more evacuations occur in the twenty-four hours, and that these are large, often putty-like, and always offensive. The nurse will insist that the child passes more than he takes. Certainly the greater part of the food he swallows is discharged undigested from the bowels.

These symptoms go on for weeks without change. The patient continues to lose flesh and seems to grow duller and paler every day. His bowels are variable. Sometimes he may have only one or two copious pasty stools. At other times the evacuations are more numerous and thinner or even watery. At times, indeed, there is actual diarrhœa, and for a few days the patient is seriously purged, the stools being frothy and sour-smelling, or thin and dark coloured like dirty water. They are always exceedingly offensive. As the complaint goes on the attacks of acute diarrhœa return more and more frequently until the bowels become persistently loose. Still, it may be months before the

stage of actual diarrhœa is reached. For a long time the child, although weakly, ailing, and spiritless, has a good appetite: indeed, often he shows great eagerness for his food. He is not feverish: on the contrary, the internal temperature of the body is often below the normal level, and his extremities are always cold. He perspires copiously at night about the body, sleeps uneasily, and is very difficult to please. There is, however, no pain; and the absence of more definite symptoms in a child whose nutrition is so obviously at fault may be a cause of much perplexity.

When diarrhœa is established the stools vary in character from time to time. They always have a horribly offensive smell and are dark coloured and watery, or drab coloured like thin paste, or very loose like chopped spinach diffused through a brownish water. In advanced cases they deposit a shreddy sediment mixed with small black clots of blood. We may then suspect ulceration of the bowel, especially if there be any tenderness of the abdomen on deep pressure.

The number of stools is subject to great variety. At times the purging abates for a time. The bowels are then moved only two or three times in the day, and the child is brighter and more easily amused. The looseness then returns, and the patient is relaxed ten or a dozen times in the twenty-four hours and again becomes dull and spiritless. The persistent diarrhœa interferes seriously with the child's nutrition, and the loss of flesh goes on quickly. The face gets hollow and lined; the eyes are dull, sunken, and tearless; the forehead is wrinkled, and a curious look of age is given by a deep furrow which passes from the nose round the corner of the mouth. The complexion is earthy, especially about the forehead, and the expression is peevish or sad. The fontanelle is deeply depressed. The abdomen is often swollen from flatulent distension, but the liver and spleen are of normal size, and no enlargement can be felt in the mesenteric glands. In this stage the perspirations usually cease, and the skin over the whole body becomes harsh and dry. The coldness of the extremities is remarkable, and the hands and feet may have a purple look; often they are swollen from œdema. The water is scanty and may contain lithates or a little uric acid sand. The child is very weak and lies quietly in his cot taking little notice of anything. If able to talk he often refuses to answer questions, or replies merely by a whine or a fretful shake of the head. His appetite may still be good, but often it is poor, so that the child can only be fed with difficulty. In this state the patient may lie for weeks, getting more and more feeble, and die at last gradually from pure exhaustion or suddenly from collapse of the lung. Sometimes death is preceded by the symptoms of "spurious hydrocephalus."

Although chronic diarrhœa in the infant usually occurs during the period of dentition, it does not necessarily bear any special relation to the appearance of the teeth. Often the teeth are cut regularly and easily without affecting the progress of the diarrhœa either for good or ill. In other cases—and this is seen especially at the later period of dentition

—the coming of each tooth is marked by a notable increase in the purging, and the looseness moderates at once when the tooth breaks through the gum. That a catarrhal derangement should be influenced by teething is no matter for surprise. When the gums are turgid and inflamed there is always some fever, and a feverish child is necessarily more sensitive than another to the ordinary causes of chill.

The frequent variations which take place in the number of the stools and the severity of the symptoms is very distressing to those interested in the patient, for their hopes are being continually raised up only to be thrown down again. After a time, however, with care, a real improvement begins, and the first sign of amendment is a change in the appearance of the stools which grow more homogeneous and solid and begin to contain bile. The child looks brighter in the face, and when he cries the eyes again become suffused and watery. This reappearance of tears is a sign of improvement of no little value. The fœtor of the stools is slow to subside. Even after the child has gained considerably in flesh and strength the stools often remain exceedingly offensive. Recovery, too, is never uninterrupted. Relapses almost always occur however carefully the child may be tended and fed. These often follow a fall in the barometer: indeed, the sensitiveness to barometric changes is so extreme that on damp days the patient is always less well than on days which are bright and dry.

In children of four or five years of age and upwards a chronic diarrhœa is more likely than in an infant to be dependent upon some constitutional cachexia. I have known a syphilitic ulcer of the intestine to keep up a looseness for many months before the nature of the lesion was detected, and in consumptive families there is often tuberculous ulceration of the bowels. In the latter case the abdominal swelling is accompanied by signs of tenderness on pressure; there is fever; the superficial veins of the abdomen are often visible; the liver is perhaps enlarged from fatty degeneration, and swollen mesenteric glands can often be detected on deep pressure in front of the spine. The nutrition of the child necessarily suffers and he gets pale and thin, but the rapidity of the wasting depends less upon the ulceration than upon the violence of the purging, the amount of fever, and the presence or absence of disease elsewhere. The prognosis, however, is serious, for the intestinal complaint is almost always only one of several manifestations of the constitutional mischief.

After recovery from a prolonged attack of chronic diarrhœa the patient remains for many months anæmic, weakly, and subject to relapse. The digestion is very poor and any—even the smallest—error in diet may be followed by a return of the derangement. This is true not only of infants, but also of children of seven or eight years of age and upwards. In the latter an occasional consequence of the complaint which ought to be mentioned is a curious check to the growth of the patient. I have seen several such cases. In one of these—a little girl who suffered for five years from repeated and prolonged attacks of chronic diarrhœa—the

child, who is now ten years old, weighs 26 lbs. 9½ ozs. and measures only 3 ft. 3¾ in. in height. During the last twelve months she has grown only ⅞ in. Another little girl—now aged eight years and a half—also suffered for several years from repeated attacks of chronic intestinal catarrh, each of some months' duration and separated by periods of comparative health. She had, I believe, at one time ulceration of the bowel. This child measures 3 ft. 6⅛ in. and weighs 2 st. 12 lbs. Both the patients look curiously small for their ages, and are only kept in good health by strict diet and constant care.

Diagnosis.—In every wasting infant the motions should be inspected as a matter of course. If this be done the nature of the complaint can hardly be overlooked. Examination of the stools is of especial importance in the early stage before diarrhœa has become confirmed. At this time the mother makes no complaint about the bowels; indeed, she rarely refers to them except to say that they are not relaxed. Careful inquiry is therefore necessary to determine the fact that of the food taken the larger part is escaping digestion altogether. When this point is settled, and we notice the normal evening temperature, the cause of the loss of flesh is no longer a mystery. Any fear, therefore, that the child is becoming the subject of tuberculosis may be laid aside.

When the diarrhœa has become constant, ulceration of the bowels may be suspected if there is moderate increase of tension of the abdominal wall, especially in the right iliac fossa, or sign of tenderness on deep pressure. The stool most characteristic of the lesion consists of an offensive dirty brown liquid containing scraps or flakes of solid matter and little black blood-clots. In infants ulceration, if it occur, is rarely due to constitutional causes, but in older children it may be of a tuberculous nature. The chest, therefore, should be examined for signs of consolidation and the abdomen for any indication of peritonitis. If these can be excluded and the evening temperature be normal, we have no grounds for suspecting the existence of constitutional mischief.

Prognosis.—So long as the purging is only occasional the child under appropriate treatment may be expected to do well. It is in cases where the looseness has become a confirmed diarrhœa that the issue is doubtful. The danger is greater in infants, and in them is serious in proportion to the degree to which general nutrition is impaired. The appearance of thrush in the mouth is a bad sign; and œdema of the extremities, as it shows marked poorness of the blood, warns us that the strength of the patient is getting low. Ulceration of the bowels in an infant is a very serious condition and likely to prove fatal. In older children the patient may still recover if well treated and nursed, provided that the lesion be not dependent upon diathetic influence. Even in the latter case I have known recovery to take place in a strumous subject in whom from the nature of the symptoms and the character of the stools I believed tuberculous ulceration of the bowels to be present.

Treatment.—To succeed in the treatment of chronic diarrhœa, we must look upon the complaint as consisting of a series of catarrhs of the

bowel due in the main to separate impressions of cold. We must take every care, therefore, to guard the sensitive body from chill, and to provide a diet which will not overtax the weakened powers of the stomach or supply material for the fermentative action of micro-organisms.

In the early stage of the complaint, when the stools consist of masses of pale, pasty matter, it is best to forbid milk in any form, and to use instead fresh whey and barley-water mixed in equal proportions with a good spoonful of Mellin's food. If the patient be an infant he may take for other meals fresh whey and cream, weak veal broth and barley-water, or the yolk of an egg beaten up with whey or veal broth. A sufficient variety should always be prescribed, and care must be taken that the whey and such like foods are in good condition, for stale whey or old barley-water is sure to disagree. The child's feet and legs must be swathed if necessary in cotton-wool, and the washing-bath must be carried out in such a way as not to chill the child.

For medicine, a few grains of prepared rhubarb may be given at night with a grain or two of aromatic chalk powder, and in the day-time half a drop of laudanum three times a day in an alkaline mixture with carminatives. This treatment will soon alter the character of the stools, and if due care be taken to protect the child from cold, milk may soon be resumed; but it is always wise to give one of the desiccated milk foods or other form of preserved milk for a few days before beginning the fresh cow's milk. As a help to digestion Finkler's papain is very useful given in doses of two grains with each bottle of food.

In the case of older children, besides forbidding milk, we must restrict the quantity of farinaceous matter. Toast or malted bread and rusks are to be preferred to ordinary baker's bread; and well-boiled vegetables, such as cauliflower, or vegetable marrow, should be substituted for potatoes. Eggs are harmless, and mutton, poultry, game, and white fish may be taken freely. Variety should be aimed at, and it is wise to provide with each meal some form of nitrogenous food, such as meat, egg, bacon, or thinly sliced ham or tongue, so that the patient may not be dependent upon toast or rusk for the means of satisfying his hunger.

If in spite of this diet the stools still continue to be pasty and large, the amount of starch should be still further reduced. A welcome help in such cases is raw or very underdone mutton pounded and rubbed through a fine wire sieve. Of this food the child should be encouraged to eat largely, and often he takes it with great relish.

At the same time that the diet is regulated, the stools must be carefully watched. If they become loose, the treatment should be that recommended for the acute attack. Offensive stools require antiseptic remedies, such as resorcin, naphthalin, or perchloride of mercury; and with each dose it is well to combine a drop or two of laudanum to quiet too energetic a peristaltic action of the bowels. Excess of mucus in the evacuations is usually stopped by the castor oil and opium mixture recommended for certain cases of inflammatory diarrhœa. In very

chronic cases, where the motions are habitually frequent and watery, nitrate of silver should always be given in doses of gr. $\frac{1}{8}$-$\frac{1}{4}$ with a drop or two of laudanum and the same quantity of dilute nitric acid three times a day.

Cases of chronic diarrhœa can usually be cured if care be taken to keep the patient warm and to protect him from every source of chill. Until he is well advanced towards recovery the washing-bath should be viewed with great suspicion. When washing is needed the patient should be placed in hot soap-suds and sponged rapidly, and no soaping outside the bath should be allowed. In bad cases, however, it is wiser, while the bowels remain loose, to forbid all washing of the child's body, with the exception of local sponging after the stools, and to keep his feet, legs, and belly thickly swathed in cotton-wool. I have in my mind not a few patients who owed their recovery, in my opinion, mainly to the use of this simple precaution.

EUSTACE SMITH.

REFERENCES

1. BALLARD. *Brit. Med. Journ.* 1883, ii. p. 363.—2. BOOKER, Dr. *Trans. of Amer. Ped. Soc.* 1889.—3. DONKIN, Dr. H. B. *Diseases of Childhood*, 1893.—4. EPSTEIN. *Prag. Med. Woch.* 1880, No. 145.—5. ESCHERICH. *Die Darmbacterien des Säuglings.* Stuttgart, 1886.—6. FINKELSTEIN. *Deut. Med. Woch.* September 17 and 24, 1896.—7. HAYEM, G. *Il Morgagni*, May 1887.—8. HOLST. *Festskrift till Prof. Herberg.* Christiania, 1895.—9. KJELLBERG. "Parenchymatöse Nephritis im frühen Kindersalter," *Journal für Kinderkrankheiten*, 1870, p. 192.—10. LESAGE. *Bulletin Med.* xxvi. p. 10, 1887.—11. LUFF, ARTHUR P. *Lancet*, 1890, vol. ii. p. 1325.—12. MARFAN. *Sem. Med.* August 21, 1896.—13. NOTHNAGEL, H. *Beiträge zur Physiologie und Pathologie des Darms.* Berlin, 1884.—14. PRIESTLEY, Dr. J. *Report for the Borough of Leicester for* 1894.—15. SEVESTRE. *Revue des Mal. de l'enfance*, March 1892.—16. SIEBERT, A. *Arch. of Pædiatrics*, 1889, p. 222.—17. TOMKINS, Dr. H. *Report for the Borough of Leicester for* 1887.—18. VAUGHAN. *Trans. of Amer. Ped. Soc.* 1890.—19. WILLIAMS, DAWSON. *Occidental Medical Times*, October, 1889.

E. S

SPRUE, OR PSILOSIS

SYNONYMS.—*Aphthoides chronica, Cacheria aphthosa, Chronic aphthæ, Chronic diarrhœa, Chronic dysentery, Diarrhœa alba, White flux, Chronic enteritis, Chronic enteritis of warm countries, Aphthæ tropicæ, Aphthæ orientalis, Stomatitis intertropica, Ceylon sore mouth, Indische spruw, Spruw, Apepsie or Athrepsie coloniale atrophique, Chronic or Endemic diarrhœa of Cochin-China, Psilosis linguæ et mucosæ intestini, etc.*

Definition.—Sprue or psilosis may be defined as an insidious, chronic, remitting inflammation of the whole or part of the mucous membrane of the alimentary canal, occurring principally in Europeans who are residing

or have resided in tropical or subtropical climates. It is characterised by irregularly alternating periods of exacerbation and comparative quiescence; a peculiar, inflamed, superficially ulcerated, exceedingly sensitive condition of the mucous membrane of the tongue and mouth; great wasting and anæmia; pale, copious, and often loose, frequent, and frothy fermenting stools; very generally by more or less diarrhœa; and also by a marked tendency to relapse. Sprue may either be primary, or it may supervene on or complicate other affections of the alimentary canal. Unless properly treated it is usually fatal.

Nomenclature.—Some explanation is due for the introduction, or rather reintroduction, into medical literature of the name under which I propose to describe this disease.

Although the first describers of the complaint recognised its peculiar characters, and gave it special names,[1] more recent writers have dropped these, and, whilst still recognising the peculiar clinical and pathological features, describe them under such loose and wide terms as "chronic diarrhœa," "chronic dysentery," "tropical diarrhœa," and so forth.

This looseness of nomenclature I regard as a distinct misfortune as well as a retrogression; for, as a consequence, the student is led to overlook or to ignore the special characters of the disease, and has his attention directed, not to the condition itself, but to merely one, and that not a constant constituent symptom of a complex pathological condition. The public, moreover, is likewise misled by such names. When the subject of this very serious disease is informed that he is suffering from chronic diarrhœa, he fails to appreciate the gravity of his position, and will not readily submit to a treatment which, to most persons, is a trying and prolonged ordeal. To tell him he is suffering from chronic diarrhœa is very much like telling the victim of cancer that he is suffering from "tumour"; he shirks and puts off a treatment which, did he but properly apprehend the danger, he would readily submit to.

The medical man, likewise, who encounters these cases for the first time is misled by these vague names, and it is only after more than one blunder and some years of experience, bought at the expense of his patients, that he begins to know the deadly nature of the complaint he is dealing with.

Lastly, the absence of a special name has led the writers of text-books and teachers to overlook a disease of which they themselves have had little experience in Europe, but which, nevertheless, is a common as well as a dangerous disease among Europeans in hot climates. Thus young medical men land in the tropics, not only without experience, but also without information or guidance in a very important matter.

For these and similar reasons, I think that the adoption of some such distinctive and catching name as "sprue" for this disease is desirable.

Etymologically the word I adopt is not very defensible, but neither

[1] Hillary (9) called it *Aphthoides chronica;* Latham (11), *Cachexia aphthosa;* Chisholm (5), *Chronic aphthæ.*

are many of the names in use in medicine—cancer, for example. At one time the word "sprue" was applied to what is now called "thrush" and "aphthæ"; for years, however, it has practically dropped out of the language, so that confusion is not likely to occur on this score. When my attention was first drawn to the subject, I found that the Dutch equivalent—"spruw"—was in universal use in Java to indicate this special form of tropical entero-colitis; and as the name had proved to be a very practical and suitable one there, I thought I could not do better than adopt it in a short notice of this disease I contributed many years ago to the *Medical Reports of the Chinese Imperial Maritime Customs*. I understand Dr. Henderson of Shanghai, recognising the desirability for a distinctive name, had also adopted this Dutch word. The name was quickly taken up by the profession and European public in China, where it is now well understood among the various foreign communities. I believe it is also becoming known in England.

In consequence of the introduction of this name it would seem that the public, at all events in the East, is gradually becoming educated into some knowledge of the disease thus indicated, and I believe that in this way the hands of the physicians concerned have been strengthened. Not only so, but the profession in general is also becoming alive to the importance of the subject, and in time our knowledge of an interesting and very important clinical symptom-group will be extended. These considerations seem to me sufficient justification for the adoption of the word "sprue," and for persevering in its use.

"Psilosis" (ψίλος, bare) is suggested by Thin in one of his valuable papers as a substitute for "sprue." It is expressive of the fact that bareness or rawness of the mucosa is a leading feature of the complaint. The word is certainly more scholarly than "sprue," but, at the same time, like "diarrhœa," it is but partially descriptive, and therefore misleading; moreover, I think it is less likely to become popular. It has the advantage of forming an euphonious adjective—psilotic, signifying bare, stripped, or raw, in which sense I shall use it of mucous membrane.

Literature.—The earliest, as it is one of the best and most graphic descriptions of what I would call the protopathic form of sprue, we owe to Hillary (1776). Since he wrote, most systematic writers on tropical diseases have dealt with the subject; I may mention Annesley, Twining, Martin, Grant, Goodeve, Donald, Moore, Chevers, Doutroulau, Corre, Kelsch, and Kiener, and many French writers. Sir J. Fayrer gives an excellent clinical account of it. Among the more recent contributions to the subject I may mention papers by myself, by Dr. C. L. van der Burg, and the well-known articles of Dr. Thin. The most exhaustive work on sprue, and one of the most recent, is that by Drs. Bertrand and Fontan, in which, in addition to much valuable original matter, there is an excellent summary of the considerable French literature on the subject.

Geographical distribution.—Although sprue is very much more common in some parts of the tropical world—Java and Cochin-China, for example—

than in others, there is good reason to believe that it occurs occasionally in most tropical countries, if not in all. Many of the cases called "chronic diarrhœa" or "chronic dysentery," coming from the West Indies, from tropical America and from Africa, answer in their descriptions to sprue. Hillary and Chisholm certainly encountered it in the West Indies. There can be no doubt about its frequency in India, Ceylon (7), the Malay Archipelago (Burg and others), the Eastern Peninsula (Bertrand and Fontan and many others), Mid-China, and especially in South China and Manila. Though most common in the tropics, it is not strictly confined to these regions; it is not infrequently met with in Shanghai, and in the European settlements on the Yangtsze River, many degrees to the north of the tropics. It may even occur in Japan; I have treated a well-marked example of the disease from that country. Although its development and rapid progress seem to be favoured by the perpetual summer and damp heat of such countries as Java and Cochin-China, yet a dry, bracing climate, such as Shanghai and the greater part of China enjoy during several months of the year, is not sufficient to ensure immunity from a disease which is apparently provoked by the great heat and relaxing influences of a long and trying summer. Speaking from my own experience in South China, where the winter climate is very fine, of all the *chronic* diseases the European has to contend against there, sprue, in its various forms and degrees, is by far the most frequent as well as the most formidable.

Etiology.—Amongst the remoter causes of sprue prolonged residence in hot climates must be reckoned as the chief. In certain instances the disease shows itself after a residence of one or two years only, or even less. This, however, in my experience, is a rare occurrence; usually I find that the victim of sprue has resided in the tropics for many years.

As more direct factors may be enumerated exhausting diseases, particularly those of the alimentary canal, such as dysentery, diarrhœa, hæmorrhoids, fistula in ano, the morning diarrhœa of the tropics—an affection which must not be confounded with the morning diarrhœa of alcoholism—child-bearing, miscarriages, and other uterine troubles, particularly those attended with hæmorrhage, prolonged lactation, syphilis, courses of mercury and iodide of potassium, bad food and water, mental anxiety, chills, and so forth. Malaria has, of course, been cited as a cause of sprue; I cannot say, however, that there is much evidence of this. Nor can it be said that bad or insufficient food is a very potent factor, for the disease is common enough in places—Shanghai, for example—where the food market cannot be surpassed; moreover, it is quite as prevalent among the rich as among the poor.

Of the actual cause of sprue nothing is known; we cannot even say whether it be of a physio-pathological character, or a specific germ. At one time the discovery by Normand of the parasite which Bavay called *Anguillula stercoralis*—since been proved to be the free form of *Rhabdonema intestinale*—in the stools of cases of Cochin-China flux, gave rise to the notion that sprue was a helminthiasis similar in some respects to ankylo-

stomiasis. This hope has been abandoned, as in many cases these parasites are entirely absent. In two cases I have searched for *Amœba coli*, but without success. Although it is possible that one of the innumerable species of bacteria present in the stools in sprue may be the cause of the disease, such a relationship has not yet been proved. It was observed by Dr. Thin that in one of his cases the stools, particularly during the exacerbations, contained almost a pure culture of a particular species of bacterium. This is a fact to be noted, but not one as yet from which to draw any definite conclusions.

In connection with the etiology of sprue it is of importance to bear in mind that whatever the actual cause may be, it is something which can remain latent for a considerable time; for one who has resided in a sprue country may return to Europe apparently in good health, and remain so for months or even for years, and yet after this interval the disease may show itself in him for the first time; or a patient may recover from the disease, and yet, without leaving England, may suffer a relapse after months or years of good health.

Symptoms.—Insidiousness, chronicity, improvement and relapse, and slow progress on the whole are characteristic of the evolution of this disease.

The leading symptoms, when it is thoroughly established, are:—

1. Irregular action of the bowels.

2. The passage of copious, pale, drab-coloured, yeasty-looking, sickly-smelling stools.

3. Tenderness, and often great soreness of the tongue, buccal mucous membrane, fauces, and sometimes of the gullet, depending on a complexity of surface lesions including (*a*) denudation of the epithelium of the mucous membrane generally; (*b*) the formation of minute herpes-like vesicles, single or in groups, with an inflamed areola, which quickly rupture, leaving (*c*) small, superficial but exquisitely tender, ashen-gray ulcers; (*d*) larger, inflamed, bare, slightly-eroded patches, smooth on the surface usually, or with a slight muco-purulent covering where in contact with the teeth—as when on the inside of the cheeks or lips; often, when on the soft palate, they are markedly granular, probably from inflamed follicles; (*e*) congestion and swelling of the fungiform papillæ, especially about the tip and edges of the tongue; (*f*) superficial cracks on the dorsum and edges of the tongue; (*g*) during complete remission of the acute symptoms, as happens occasionally in most cases, an atrophied state of the entire body of the tongue; this organ then appears pale and almost cartilaginous, with a smooth, glazed surface as if coated with varnish, and completely denuded of papillæ.

The erosions referred to, sometimes amounting to superficial ulceration and much sodden by the constant action of the saliva, are most frequently found under the tongue by the sides of the frænum, inside the lips and cheeks; especially where in contact with the teeth, and on the soft palate. The vesiculations ending in the minute gray ulcers are commonest about the tip and edges of the tongue; they come out in

successive crops. Activity of both of these lesions is usually associated with increase of diarrhœa. In severe cases what I would call the "psilotic" condition of mucous membrane seems to extend through the entire length of the alimentary canal, affecting the pharynx, œsophagus, the anus, and, in women, even the vagina.

4. Dyspeptic conditions associated with the formation of large quantities of intestinal gas and acidity.

5. Pearly conjunctivæ; a dry, harsh, earthy-looking skin more or less deeply pigmented.

6. General wasting, usually including marked diminution of the area of hepatic dulness.

7. Anæmia and great physical and intellectual debility; and, especially in the advanced stage, querulousness and irritability of temper.

If we inquire into the history of a patient presenting these symptoms, we shall probably learn that he has been ailing for several months, perhaps for years; and that his symptoms began in one of three ways:—(*a*) Insidiously: he will tell us that before his disease definitely declared itself he used, without apparent reason, to be troubled with soreness of the mouth, attacks of flatulent dyspepsia, irregularity of the bowels, and, especially, a tendency to diarrhœa—often of pale, copious, frothy stools—in the early morning or during the forenoon. By degrees such attacks became more frequent, the mouth oftener sore, and the dyspepsia more troublesome. Ultimately this state became permanent, his strength and flesh began to decline, and, at last, a condition of confirmed invalidism was established. (*b*) Suddenly: being at the time in apparently good health—possibly after exposure, chill, fatigue, bad water bad food, dietetic excess, or some other indiscretion—the patient was suddenly seized with profuse diarrhœa. After a time, the urgency of the diarrhœa subsiding, a degree of looseness and irritability of the bowels was left and became permanent, the stools gradually losing their yellow or brown colour and becoming pale and fermenting; and the tongue, from being thickly coated, became at first abnormally clean with a liability to the formation round the edges and tip of small painful ulcers, and, ultimately, this organ taking on the psilotic character, the other symptoms of confirmed sprue were gradually evolved. (*c*) Secondarily: the patient had suffered from well-marked dysentery, which had merged into the chronic form, attended with the passage of three or four ill-smelling, mucoid, slightly bloody stools daily. After several months or, perhaps, a year or two of this, the mouth began to lose its natural fur, and to be bare or tender; flatulent dyspepsia with much distension after eating became troublesome; the stools got more massive, paler, yeasty-looking; and, ultimately, this condition becoming chronic, the dysenteric element gradually ceased to be the principal feature in the clinical picture, giving place to the characteristic symptoms of sprue.

These I consider to be the principal types of sprue. There are others, doubtless; the various combinations and degrees of severity and rapidity in the development of the leading symptoms are infinite. From what I

can gather, I consider that in China the form of sprue most frequently met with is the protopathic form; in Java and Manila the same seems to be the case; but in Cochin-China and in India it would seem that in a large proportion of instances the disease is secondary to acute catarrhal enteritis, to dysentery, or to the affection called "hill-diarrhœa."

Forms of imperfectly developed or nascent sprue are common enough in the tropics. Sore mouth, with or without morning diarrhœa (sometimes of bilious stools, sometimes of pale fermenting stools), is a common affection among foreigners in the East, and, if ordinary prudence be exercised, may not go further or seriously affect the health. Sometimes indeed, tenderness of the mouth, burning of the tongue or gullet when sapid, alcoholic or pungent foods or drinks are taken, or, perhaps, minute blisters followed by equally minute aphthous ulcers giving rise to actual soreness of the mouth, are the only symptoms present, even in cases with cachectic features. I once watched a case of this description during several years. The patient had resided for many years in the tropics, and on the whole had enjoyed good health. The only serious exception to this was an attack of dysentery many years before the appearance of the troubles for which I was consulted. Ever since the dysentery, however, attacks of sore mouth had supervened from time to time, being induced more particularly by nervous shock or similar physiological strains. The last attack had been unusually severe and protracted, and was accompanied by great wasting of a semi-cachectic kind, but there was no diarrhœa. It was only by careful dieting during many months that the psilotic condition of the mouth, and probably of the stomach and upper part of the alimentary canal, was overcome, and the general state of the nutrition improved. Had this case been neglected or wrongly treated, doubtless, in a very few months, diarrhœa of pasty, fermenting stools would have supervened and ultimately passed into confirmed sprue.

Just as in this case, although the tongue lesion was very well marked, diarrhœa was at no time a feature of the complaint; so in others, as pointed out by Bertrand and Fontan, diarrhœa and wasting of a characteristic type may exist, and yet the tongue retain its epithelium and be free from the erosions and inflamed patches so typical of sprue. Indeed, these writers seem inclined to minimise the significance of the psilotic condition of the mucous membrane of the mouth as an index of the condition of the remainder of the alimentary canal. It must be admitted, however, that the morbid process in this disease may be patchy in its distribution, and that it may be confined to the lower part of the bowel; the higher part—of which the tongue more particularly serves as an index—remaining healthy.

In many cases the soreness of the mouth is exceedingly troublesome; only the very blandest fluids can be tolerated. Even the movements of deglutition may be excessively painful, yet they are constantly provoked by the salivation induced by the state of the mouth. In other cases the mouth—especially during sleep—becomes very dry, the tongue literally so cleaving to the roof that to get it moistened and liberated every

morning is quite an operation. Many complain bitterly of a feeling of soreness or burning, doubtless depending on erosion of the œsophageal mucous membrane, which they locate beneath the sternum. Hunger, too, sometimes amounts to acute suffering, and, as with diabetics, may lead to strange moral perversions. I have seen cases in which the constant craving for food brought on a hysterical condition in which the patient, provoked by some savoury smell and throwing prudence to the winds, rushed to table and indulged indiscriminately in the family meal. Thirst, too, may be a troublesome symptom. So may be distension of the abdomen from flatus and accumulated food; a smart attack of diarrhœa affords welcome relief in such cases. Sleeplessness is often complained of. In sprue the temperature is below normal, and the patient's feet are often painfully cold; just before and during an exacerbation of symptoms there may be a rise of one or two degrees in the body temperature, at other times it is subnormal. As the disease progresses both the number and value of the red blood corpuscles diminish, the relative proportion of the white increasing; as recovery sets in this, of course, is reversed. The quantity of urine passed is in inverse proportion to the diarrhœa.

When sprue has become thoroughly established, unless a proper course of treatment be adopted and persevered in for months or perhaps years, the disease as a rule becomes gradually worse,—slowly, it may be, but on the whole none the less surely. Temporary improvements are followed by relapses; the slightest dietetic indulgence, exposure to wet or cold, fatigue, mental depression, and such like occurrences, are nearly sure to bring about a fresh attack of diarrhœa and an aggravation of the mouth symptoms. So the case gradually goes from bad to worse, emaciation becomes extreme, and the dusky, dry, unhealthy, earthy-looking skin hangs in loose folds on a skeleton every bone of which can be counted. The belly alone is filled out; it is generally ballooned by accumulated gases, and the gurgling and movement of gas and fluid can be seen, heard and felt. The liver, in harmony with the wasting of the rest of the body, is shrunk to abnormally small dimensions. Sometimes a trace of albumen may appear in the urine. Finally the mouth becomes covered with mycotic thrush, feet and ankles swell, and the patient, if not suddenly killed by an attack of choleraic-like diarrhœa or other intercurrent acute disease, dies slowly of starvation.

Should a proper treatment be adopted in time and before the mucous membrane has been so extensively destroyed as to be unfit for digestion, and should the patient have not yet entered upon the vicious pathological circle to be presently described, the diarrhœa will cease, and the psilotic state of the mouth subside, to recur in milder form and at longer intervals until it disappears for good, and the stools will gradually regain their proper colour—at first, perhaps, in a fitful sort of way, one part of a motion being properly coloured whilst the remainder is still pale. By degrees the patient gains flesh, and, finally, when he recovers the power of digesting a fairly liberal diet, his muscular and intellectual powers will

be restored. For a long time, however, looseness of the bowels, slightly sore mouth, and flatulent dyspepsia may recur on the smallest provocation —symptoms showing that the disease, although apparently cured, is only slumbering.

Occasionally it happens that careful treatment, by checking the more urgent symptoms, gives rise to a deceptive arrest of the disease; the soreness of the mouth ceases to recur, and diarrhœa is in abeyance. Notwithstanding this, nutrition does not improve; the food the patient takes adds nothing to his weight or strength, both of which gradually decline, without any very manifest reason, as in diseases like cancer or pernicious anæmia. In all such cases it will be found that the stools are abnormally large. Doubtless too large an extent of mucous surface has been irretrievably destroyed, and absorption does not balance waste. In such a case the right treatment has come too late.

Pathology and pathological anatomy.—Owing to practical difficulties in examining a structure so delicate as the mucous membrane of the alimentary canal, and to the fact that in a disease so chronic the primary and essential features of the complaint become overgrown and masked by secondary changes, it has hitherto been found impossible to ascertain the primary and fundamental lesion of sprue. It seems to me that more information is to be got on this point from a study of the lesions of the tongue and mouth in the early stages than from the study of the lesions of the other parts of the alimentary canal in the later stages of the disease. If the tongue be a trustworthy index of what is going on in other and lower parts of the alimentary canal, then we must conclude that the primary lesion, at all events the one first appreciable by the unaided senses, is a catarrhal state of the mucous membrane as evidenced by premature shedding of the epithelial covering, congestion of the papillæ, and folliculitis going on to superficial ulceration. What in its turn may be the cause of this catarrh we cannot say.

Whether it be in consequence of this hidden, unknown primary cause, or of the catarrh which it induces, it is very certain that in sprue digestion and assimilation are early affected; and that this, in no very long time, leads to tissue starvation. To this tissue starvation, doubtless, is attributable not only much of the general wasting but many of the special symptoms referable to the alimentary canal. Dr. D. D. Cunningham, in a very able paper on the effects of starvation on vegetable and animal tissues (6), has shown that the effect of starvation in Indian famines is not only to cause a loss of bulk of the tissues generally, but also, if the starvation be carried beyond a certain point, to actual destruction of tissue. Particularly is this the case with the mucous membrane of the alimentary canal, in which, in famine, an actual and irreparable destruction of secreting and absorbing tissue is apt to occur. As a result of this destruction, when sufficient food is at last supplied, as in famine camps, digestion and absorption, owing to actual loss of the necessary tissues, cannot be effected; thus, so far from proving a benefit to the starving, good food and plenty of it does but precipitate death,

inducing by its mechanical and chemical effects further irritative changes in the starved and eroded bowel. In this way is brought about the diarrhœa and dysentery of famine camps, and many of the diarrhœas and dysenteries of the half-starved populations of Eastern countries who, in too many instances, live in a state of chronic famine—a state hardly conceivable by the fortunate populations of more favoured countries.

Doubtless something analogous to this famine diarrhœa and dysentery occurs in sprue ; an artificial physiological famine is induced. Therefore, in contemplating the lesions found post-mortem in a case of sprue, we must be careful to bear in mind that many, if not most of them, are not the specific and primary lesions of sprue, but the primary and secondary effects of starvation. Thus, when at the end of a long case the alimentary canal is examined, there is present a threefold pathological condition, namely—

(*a*) The specific and primary lesion,
(*b*) The specific starvation lesion,
(*c*) The secondary irritative lesions.

To sort out these in the present state of pathological knowledge would be an impossible task. Very few post-mortem examinations have been recorded by recent English observers with the thoroughness and minuteness of histological detail necessary to enable us to carry out such an attempt successfully. Indeed, the only case I know of in very recent English medical literature available for the purpose is one recorded by Drs. Thin and Wethered (14). The French have done very much more in this direction, but even their elaborate investigations seem to be principally illustrative of the secondary and tertiary starvation changes, and not of the primary and essential lesion.

After death from sprue the tissues as a whole are found to be abnormally dry, in consequence of which the body tends to mummify rather than to decompose. The thoracic and abdominal viscera are both actually and relatively wasted. Tubercular deposits may be found in the lungs. In the liver there are no morbid appearances which, from their invariable presence, might be considered essential. More frequently, according to Bertrand and Fontan, certain peculiar and hitherto undescribed changes are encountered in the pancreas, isolated lobules of which show signs of parenchymatous change—such as fatty or granular degeneration of the cells, with softening of the acini and slight inflammatory infiltration of the connective tissue. The pancreatic changes, however, are probably secondary and not essential, as, like those sometimes found in the liver and also in the kidneys, they are far from being constantly present.

As might be expected, the principal changes are to be found in the alimentary canal, part or the whole of which, including in many cases the stomach, is invariably found to be diseased. Speaking generally, the bowel may be described as being thinned to such an extent as to be diaphanous. There is no necessary or notable lesion of the serous or

muscular coats; but from mouth to anus, either in segments (in Thin case the œsophagus and ileum only, the remainder being healthy), or in patches of greater or less extent, or universally (Bertrand and Fontan the mucous membrane is diseased. Its free surface is coated with a thick layer of dirty, viscid, gray, tenacious mucus. On washing this away the mucous membrane is found to present a variety of lesions:—1st, congested patches; 2nd, eroded patches; 3rd, ulcerated patches; 4th, pigmented patches; 5th, bare, thin-scarred, cicatricial patches; 6th, absence of, or wasting of villi and glands; 7th, in addition to these the mucous membrane, where not entirely replaced by fibro-cellular new scar growth is felt and seen to be sown with minute spherical tumours—about the size of a pin's head—surrounded by a dark pigmented areola. These little nodules, on being cut into, are found to be situated in the submucosa, to have gelatinous contents, and generally a lateral or central punctiform orifice. They are mucous cysts replacing follicles (10).

Under the microscope, sections of the diseased portions of the œsophagus, stomach, and intestines show patchy or general destruction of the surface of the mucosa in all degrees from slight erosion to complete disintegration of the villi, glands, and follicles; effusion into and around the closed follicles, leading to the formation of mucous cysts or of small abscesses which subsequently rupture and ulcerate; infiltration by leucocytes of the basement membrane; and inflammatory effusion into the submucous layer, with subsequent fibro-cirrhotic changes in the new tissue.

The mesenteric glands also are generally enlarged, dark, and perhaps fibrotic.

The ulceration met with is not always extensive. In the stomach and upper parts of the alimentary canal the sores are generally very minute and superficial; towards the end of the ileum and in the colon the ulcers may be larger, deeper, and associated with some thickening of the bowel from inflammatory effusion.

Speaking of the changes in the stomach, Bertrand and Fontan say "It may be completely destroyed by embryonic cell proliferation in the mucosa on the one hand, and by ulcerative folliculitis on the other. Finally it presents sclerosed and pigmented portions which are probably the ultimate stage of the nutritive and circulatory troubles." Dr. Thin remarks of the ileum in his case, "The mucosa was almost entirely destroyed, being replaced by a structureless substance enclosing leucocytes and here and there the remains of a follicle. . . . The submucosa appeared much thickened, and the fibrous tissue abundant and more than usually solid in consistence. The walls of the vessels were also thickened altogether denoting a more chronic process than in the other parts of the intestinal canal."

If I may venture to interpret these morbid appearances I will suggest that, as the primary lesion in the alimentary canal in sprue is one from which recovery is possible, and as most of the lesions of the stomach and ileum just described are of such a character that recovery from them i

impossible, these latter may be regarded as a result, but not the necessary result of the primary lesion—not the essential features of the disease. The primary pathological condition of the mucous membrane, seeing that it admits of recovery, is probably of the nature of a catarrh, and confined to premature shedding of epithelium and folliculitis; this view is further borne out by what is seen in the mouth. After a time, this condition being unrelieved, more serious changes may supervene; the necrotic famine changes, or inflammatory changes induced by persistent irritation, may set in. Either or both of these may lead to ulceration on the one hand or to cirrhotic changes on the other; and in one or both of these ways the mucous membrane is changed into scar tissue without glands or absorbents, gradually becoming perfectly useless as an agent in nutrition.

Applying the revelation of the post-mortem room to the interpretation of symptoms, we can understand that according to the region and extent of the alimentary canal involved will the symptoms be. If the œsophagus be implicated, dysphagia and substernal burning will be prominent symptoms; if the stomach be involved, vomiting and dyspeptic troubles of various kinds will occur; if the intestines, lienteric diarrhœa; if the colon, dysenteric diarrhœa. All of these conditions and symptoms may and often do combine in one and the same case.

The frequent presence of ulcers in the colon has given rise to much discussion, many French observers contending that they are conclusive evidence of the dysenteric nature and origin of sprue. But it is evident from a study of the history of the evolution of this disease that, in a large proportion of cases, the progress of the pathological process is from mouth to colon, and not from colon to mouth. The psilotic process is a distinctly different one from the dysenteric process, although they may and do concur. True dysentery may in some instances be the starting-point of sprue, just as the damage inflicted by an antecedent pleurisy may favour tubercular deposit in a lung; but sprue is not true dysentery, any more than phthisis is pleurisy. And as phthisis may give rise to pleurisy, so may sprue give rise to a kind of dysentery—meaning by dysentery simply ulceration of the colon; or even, by weakening the resistive power of the mucosa of the large intestine, dispose to true dysentery; the presence of tubercle near the pleura accounting for the association in the one case, and the frequent passage of irritating discharge accounting for the association in the other.

An interesting feature in sprue is the absence of the normal yellow tint in the stools. Several hypotheses have been put forward to explain this, but it cannot be said that any of them has been proved. The physiology of the formation of the colouring matter of the healthy stool is by no means settled; until this is made out, any theory about the cause of its disappearance in sprue must be in the highest degree hypothetical. Thin suggests that in sprue the normal colouring matter of the stool is destroyed by a bacterium whose presence is favoured by the abnormal acidity of the contents of the bowel, the consequence of deficiency of

alkaline secretion. Bertrand and Fontan's observations on the implication of the pancreas might have a bearing on this question, seeing that hydrobilirubin, the cause of the colour of healthy fæces, according to Walker, cannot be produced from the bile without the aid of the secretion of the pancreas. Thin's view depends on the presumption — founded on an analysis by Wynter Blyth—that the ordinary elements of the bile are present in the fæces in sprue. Bertrand and Fontan, however, in a similar analysis, failed to find the bile acids.

Of the many hypotheses which have been advanced to account for the absence of colour in the stools, the most simple, and to my mind the most plausible, is that which accounts for the phenomenon by assuming that in sprue there is a suspension, partial or complete, of the chologenetic function of the liver. Such a supposition would also explain the yeasty, fermenting state of the stools so characteristic of the disease. More facts, however, are wanted before any reliable conclusion can be arrived at on this point.

Pending further investigation I regard sprue, etiologically, as a disease resulting from a slowly-developed pathological action of meteorological conditions present in hot countries on constitutions unsuited to contend with certain more particular and local influences as yet unknown; further, I believe that these influences in many instances are of themselves sufficient to bring about the disease, but that their action is favoured by antecedent pathological conditions, over and above those induced by climate simply; chief among these are dysentery, catarrhal diarrhœa, hepatic disturbances, and other morbid states, more particularly those connected with the alimentary canal. Pathologically, I regard sprue as a morbid process specifically distinct from true dysentery, and consisting in a suspension of the chologenetic function of the liver, together with a specific, chronic, catarrhal condition of all or part of the digestive tract which, if it persist, leads in time to necrotic and inflammatory processes in the mucosa ending in destruction and permanent loss of glandular and absorbent structures, and so to what in a certain sense might be called physiological starvation.

Treatment.—This is mainly dietetic and hygienic. If carried out intelligently by the physician and conscientiously by the patient, and if instituted at a sufficiently early period of the disease, it is generally marvellously successful. On the other hand, if treatment be wrongly directed, carried out in a half-hearted way, or too long postponed, recovery becomes an impossibility.

In prescribing a treatment for a case of sprue, the pathological condition of the bowel must always be present to the physician's mind; he must never forget the eroded and tender mucous membrane, the deficiency of glandular structure and villi, and the liability of food in excess of the digestive and absorbing capacity to decompose and irritate. The indications demanded by these things are clear and distinct. They are, firstly, to place the inflamed gut, so far as is possible, in a state of physiological rest; secondly, to supply a food of as bland a nature as possible; and,

thirdly, a quantity of food no greater than can be disposed of by the glands and absorbing apparatus. For the fulfilment of these indications what is known as the "milk treatment" is incomparably the best.

When a diagnosis of sprue has been arrived at, it becomes the duty of the physician to endeavour to secure the co-operation of his patient by impressing upon him the great danger of procrastination, the deadly and insidious nature of the malady if neglected, the comparative certainty of recovery if promptly attended to. The conviction of the patient in this sense, and his or her hearty and intelligent co-operation, are the first and most necessary steps towards a cure. Few, unless they have had actual experience of these cases, can understand the importance of the moral element in their management. Next in importance to this is the acquisition of a thorough knowledge of what the patient's alimentary canal can and cannot do. This knowledge can be gained only by a daily visit to the patient, and a daily inspection of the stools; and these daily visits and inspections should be kept up until the slow process of regeneration of mucous membrane has well set in, and the patient has become thoroughly educated in his own management. There must be no temporising, and in the event of relapse, no matter how trifling apparently in character, there ought to be no hesitation in harking back at once to special diet. To some readers it may seem a matter of supererogation to write of these things; but they are really, though so simple and self-evident, among those simple and self-evident things which, though of prime importance, are so generally overlooked and neglected.

When a patient has consented to undergo the milk treatment for sprue he ought to have clear directions how this is to be carried out. These directions are best given in writing, somewhat as follows:—

1st, Clothe warmly in flannel. If diarrhœa be active go to bed in a warm room, and remain in bed until the stools become solid.

2nd, Place a pad of cotton over the abdomen, keeping it in position by a broad flannel bandage somewhat firmly applied.

3rd, Stop all food and drink with the exception of three pints of milk per diem, the milk being taken in divided doses at intervals of one hour, allowance being made for a reasonable amount of undisturbed sleep.

4th, The milk, warmed in cold weather, must not be drunk, but sipped with a teaspoon or sucked from a feeding-bottle.

It is generally desirable to commence the treatment with a teaspoonful of castor oil, and to repeat this for a time about once or twice a week, or whenever the stools become copious, frothy, or stinking.

As a consequence of this treatment, even in cases in which diarrhœa has been present for months or even years, the motions gradually diminish in number and amount, and increase in consistency. Usually before a week has passed they become solid; at the same time the soreness of the mouth diminishes, and the feelings of dyspeptic discomfort vanish. If, however, after two or three days there be no improvement in the diarrhœa, the quantity of milk must be diminished by ten ounces a day

until a minimum of 25 ounces is taken. Of course this is starvation diet. it must not be persisted in too long, and the patient must be kept a absolute rest, and carefully watched and tended by an intelligent nurse.

After the motions have become solid, and have so remained for three or four days, the allowance of milk is increased by 10 ounces daily until 100 to 150 ounces are consumed. Should the motions at any time become loose, this must be looked upon as an indication that more milk is being swallowed than can be disposed of; the daily quantity must therefore be correspondingly reduced, by half a pint at a time, until the stools again become solid. It should then, after three or four days be cautiously increased once more.

By following this system, in nine cases out of ten the improvement is marvellous. Not only does the diarrhœa cease, but the dyspeptic troubles subside, the soreness of the mouth and gullet disappears, and the patient's condition improves in every respect. There may even be a gain in weight when the allowance of milk has risen to five or six pints. But, arrived at this stage, the troubles of the physician begin. The patient is very hungry and clamours incessantly for more substantial food. The friends, too, importune on his behalf, and often do not hesitate to accuse the physician of starving his patient. All this must be steadfastly resisted, however, and for at least a month—better for six weeks—the only answer to the incessant demands for more nourishment must be "Milk only — nothing but milk." If the physician be so weak as to concede something prematurely he is pretty sure to regret it. Additional diet too soon begun means recurrence of diarrhœa, sore mouth, renewed wasting, loss of time, and, perhaps, loss of the last chance of recovery.

Experience, and one or two trials carefully made, will tell us when to begin to improve the diet. An egg beaten up with milk may be tried in the first instance, and, if found to agree, made a permanent addition to the daily allowance of milk. Then some well-boiled arrow root or corn starch, or plain rusks, or plain biscuits, or some other simple form of farinaceous food may be carefully introduced. Bread is not well borne unless in the shape of bread and milk. Bananas I have found to be well borne, and are often a very acceptable addition to the dietary; they must be given tentatively at first, and only in very small quantity and be thoroughly masticated. If they are well borne, after a time as many as five or six may be taken daily. Then chicken broth may be added in small quantities; afterwards fish, chicken, potatoes, sweet-bread and so on. Beef and mutton, hot soups, fibrous vegetables, nuts, pastry, etc. are very badly borne, even for years after all symptoms of sprue have subsided, and it is frequently necessary in such patients to interdict their use altogether.

During convalescence, and for a long time thereafter, the patient ought to be instructed that on the slightest recurrence of sore mouth or of the familiar feeling of dyspeptic discomfort, or of bulky, stinking stools, he should at once act on the hint, take a teaspoonful dose of castor

oil or of compound rhubarb powder, starve for some hours, and then go on milk for a day or two, until the threatening attack of muco-enteritis has quite subsided. He can then resume his former diet. Prompt treatment of this sort on the threat of relapse saves weeks of discomfort, and perhaps averts a serious recurrence of illness.

Until the patient is taking at least a hundred ounces of milk a day he ought to keep his bed. Afterwards, when he gets up, he must be careful to clothe very warmly, to avoid cold, damp, fatigue, excitement, and everything leading to physiological strain of any description. Getting wet or feeling cold is most prejudicial to such patients; accordingly, when the winter comes round, he should, if possible, move to some warm and sheltered place in the south of England or to the Riviera.

As regards drugs, my own experience has been that they are very secondary matters, and, with the exceptions I have mentioned, may generally be dispensed with. If there are special indications, such as malarial fever or cachexia, appropriate drugs, such as quinine, may be demanded. When diarrhœa is very brisk, so that it is to be presumed that the violent peristalsis of the gut is interfering with any healing process that may be going on, then a few small doses of laudanum or of Dover's powder may be given; but it is a very great mistake to try to shut up the bowel altogether by sedatives or astringents. The gut should be regarded in the light of a surgical drainage-tube to be kept patent; the retention of a mass of putrefying material in the abdomen cannot possibly be of any advantage in the long run, or in any way conduce to recovery.

It sometimes happens that after a week or two of careful milk dieting, the stools, although diminished in number, fluidity, and quantity, are still deficient in consistency and unformed. Diarrhœa, in the usual sense of the word, has ceased; but the complete dehydration of the motions by the last part of the alimentary canal is not thoroughly effected; apparently there still remains a mild chronic catarrh of the colon. In this case a dose of laudanum is sometimes followed by solid stools. If this fail I recommend the injection per anum through a long tube (after washing out the bowel with warm water) of forty ounces of nitrate of silver solution—one-half to one grain to the ounce of tepid distilled water. This is often followed by solid stools. It is of no use, however, to give laudanum or to wash out the bowel in this way until there has been at least a fortnight of careful dieting. When the stools are very loose and watery it does no good; properly timed, these measures are very often successful.

Much suffering is frequently caused by the raw state of the mouth. To mitigate this as much as possible the mouth should be rinsed out, every time after taking milk, with water or a weak solution of borax. Touching the painful spots with a 5 per cent solution of cocaine, or with solid nitrate of silver or sulphate of copper, gives relief. I have found the following mouth-wash useful in such circumstances:—Ol. creosoti gtt. 8, Tinct. pyrethri drs. 4, Tinct. myrrhæ drs. 2, Tinct. krameriæ

drs. 2, Aq. camphoræ to ounces 8; a teaspoonful in one or two wine-glassfuls of water.

When patients must have a mixture, the following, recommended by Dr. Thin, may be prescribed:—Magnes. sulph. grs. 8, Tinct. rhei min. 10, Syrup. zingib. min. 10, Aq. dr. 1, to be taken three times a day. I have seen no good but rather the reverse from bismuth, oxide of zinc, acetate of lead, nitrate of silver, sulphate of copper, and the vegetable astringents and similar remedies designed to act as sedatives or astringents. It is possible that carbolic acid, salol, naphthol, and similar antiseptics may be of service, but I have no personal experience of their use in these cases. When diarrhœa has ceased, nux vomica or very minute doses of arsenic, or some non-irritating preparation of iron, may be exhibited with advantage. Fresh bael fruit is greatly extolled by Fayrer, and is much employed in India in these and kindred cases. I have seen no benefit from the officinal extract.

Constipation should be systematically relieved by enemas of warm water, or, better, of linseed tea, or weak barley or rice water.

In cases of dysenteric origin, after milk diet has been thoroughly carried out for a fortnight with partial success, nitrate of silver enemas as just described, may be required. Sometimes in such cases I have found benefit from decoction of simaruba.[1] In the debilitated condition of sprue patients ipecacuanha is not to be recommended.

Alcoholic stimulants, as a rule, do harm. If circumstances seem to make their use desirable they may be given well diluted, their effects on the motions being carefully watched.

It sometimes happens that although the digestive powers are equal to the disposal of 50 or 60 ounces of milk per diem, they are unable to cope with a larger quantity, a few additional ounces bringing on diarrhœa. In such cases nutrient enemas should for a time supplement feeding by the mouth; indeed, in every case in which the rectum tolerates their presence nutrient enemas may be given with advantage. Sometimes only a very small and utterly inadequate amount of milk can be borne. In some of these cases aerated milk (3) or koumiss agrees better. There is also a class of case in which the power to absorb fluid in any larger quantity is in abeyance; in these, by thickening a small

[1] This drug has for many years been neglected in the treatment of dysentery, at all events by English practitioners. It undoubtedly possesses anti-dysenteric virtues of a high order, and is used very extensively and successfully by irregular practitioners in the East in the chronic forms of this disease. The officinal preparation is much too weak and the dose too small. In the East much larger doses are used. One method of preparing and using it which I have seen in vogue is as follows:—Simaruba, 2 ounces, divide into four equal parts, simmer one portion in an earthenware pot in a pint and a half of water for three hours, strain. Let the patient remain in bed and drink this decoction on an empty stomach every second morning for four times. The food must be very plain and consist principally of milk and farinaceous stuffs. Another method of preparation is to boil an ounce of simaruba in twelve ounces of water till reduced to seven drachms, strain; to this is added one drachm of spirit. Of this preparation—which is made in an enamelled vessel—a child may take one-fourth part, an adult the whole. The dose should be taken every night for four nights.

quantity of fresh milk with condensed milk or some form of desiccated milk, or by evaporating in shallow dishes and at a low temperature a portion of the water in fresh milk, the requisite amount of nourishment may be introduced without increasing the bulk of fluid.

Before leaving the subject of the milk treatment of sprue I would repeat that it must be thorough. I have seen cases in which the addition of a biscuit or a rusk, or worse than these, of bread, interfered with its success, and in which the withdrawal of these "extras" was very speedily followed by solid motions and improved mouth. I would also impress upon my readers the importance of securing a supply of pure milk for these patients. A little lime-water, or bicarbonate of soda, or a pinch of table salt, may be added to the milk with advantage; but such substances as boracic acid—so frequently employed in London by dealers in milk during hot weather—are exceedingly dangerous. I once saw a case nearly lost in this way. If milk is to be the only food no trouble should be spared to see that it is thoroughly good.

If after a thorough trial of milk, say for a fortnight, there is no sign of improvement, what is to be done? As regards feeding, experiment must be made in one direction after another to try whether haply some food or combination of foods can be found which will agree. Peptonised food, malted foods, or some of the many preparations of this description in the market may be tried. Fresh meat juice, scraped beef, white of egg, pounded chicken, thoroughly cooked arrow-root, corn starch, decoction of scorched rice, have all been of service; but my experience on the whole has been, that where the milk treatment has been thoroughly carried out and has failed no other treatment has succeeded.

Dr. Neil Macleod of Shanghai has favoured me with the following note on the treatment of certain cases of sprue:—

It is unquestionably the physician's first duty to resort to milk—milk alone—in dealing with this disease; in nine cases out of ten, if it is not too far advanced, a successful result will be obtained. There are, however, certain cases where with milk, though tried in various forms, the stools continue copious, loose, and offensive, and the patient will insist, and it would appear as if it were the case, that the milk makes matters worse. Flatulent abdominal distension is complained of and discomfort is obvious. In such cases a complete withdrawal of milk and the substitution of fresh beef-juice is well worthy of a trial. The quantity obtained by squeezing one pound of good beef-steak, cleared of fat and underdone, should be given every two hours for seven or eight doses daily. The addition of salt and a very little pepper renders it more palatable. Here, as with the milk, a careful watch of the daily quantity and quality of the stools is absolutely necessary to judge of the effect of treatment. Within forty-eight hours the distension and discomfort will have disappeared. The stools, now become black, will be watery for a time with minute shreds dispersed throughout. If improvement continues the shreds will disappear and, in favourable cases, the stool may be of the consistence of thin treacle, becoming gradually thicker, till in the second week a porridgy consistency may be attained. If the patient feels hungry and dissatisfied with the

juice, a half-slice of crisp toast, thin as thick cardboard, may be given with each dose, in which case the stools will be brown instead of black. The toast should be given only if the patient desires it, and this is usually the case. On this diet, with water to assuage thirst, occasionally a formed stool may occur. At the end of the second week a piece of under-done beef-steak free from fat may be given and must be well masticated. If the stool keep its favourable character as to consistence and quantity, a mutton chop without the fat may be substituted. After a few days an egg or bread and butter may be added. One thing at a time only should be added, and the result tested by watching the effect on the consistence and quantity of the stools. As in the case of the milk there must be no hesitation in harking back when indications present themselves, these indications being thinning of stools, increase of quantity, and specially offensive odour, or any of these. Starchy puddings may next be added, and having got so far a cup or two of milk daily may now be tried. It must be remembered that meat stools are more offensive than milk ones.

If every effort to check the diarrhœa or to improve the assimilation be defeated, perhaps it is the wisest course to accept the inevitable, and to prescribe a diet and a treatment most in keeping with the wishes and comfort of the patient. We occasionally read of wonderful recoveries on a mixed diet even of coarse fare. It is sometimes possible to continue the milk diet too long, and it may happen that after a time a certain freedom in the matter of food will be followed by improvement.

Recently Dr. Begg (1), who appears to have seen much of this disease in Hankow, China, claims to have found in santonine almost a specific for sprue. He places the patient in bed, and, after a dose of castor oil and, if necessary, a large enema of warm water, gives five grain doses of yellow santonine dissolved in a teaspoonful of olive oil every night or morning for six times. He insists that the santonine shall be yellow santonine; white santonine he has found to be less effectual. The diet, at first of milk, is gradually improved as the powers of digestion return. He claims that by this treatment he has cured many cases in which the milk treatment had failed, and also, he says, the yellow santonine treatment has the further advantage of being more permanent in its results and to occupy a much shorter time.

There is another system of treatment which deserves further investigation. It is what is known as the "fruit cure" or "grape cure." Van der Burg recommends it very strongly. In carrying out this system of treatment the diet must consist entirely of fruit, such fruits being selected as are pulpy and free from coarse seeds, fibres, and excessive acidity. Fresh or tinned apples, pears, peaches, apricots, strawberries, grapes, cucumbers, mulberries, mangosteens, lichjs, lungans, rambutans, melons, gourds, are some of the fruits Van der Burg recommends. The juice of oranges and pumelos he permits, but pine-apples he interdicts. I have never employed this treatment, but Van der Burg, who seems to have had a very large experience of sprue in Java, is enthusiastic in its praise, and cites several cases in which it proved highly successful. He says: "I need hardly say that some courage is

required to enter on this course, especially when diarrhœa is a prominent symptom; but the good results obtained by others and myself amply justify the treatment." I have attempted the "fruit cure" so far as to give bananas and apples liberally in conjunction with milk after the diarrhœa had ceased under the exclusive use of the latter, and with great comfort and, I believe, advantage to my patients.

The subjects of well-marked sprue in the tropics ought to be sent to Europe as soon as possible, and should not return until they have passed at least one hot season in a cool climate, or until their symptoms are thoroughly in abeyance, and they can partake of ordinary food with impunity, and are in a satisfactory state of nutrition.

Before sending the patient on board ship an effort ought to be made to check the more active symptoms; on no account should a patient be sent to endure the miseries of a long sea-voyage when in a dying condition. Before being shipped careful instructions as to diet should be given, and the patient educated in the use of milk; preserved milk can always be procured on board ship, and makes a very fair substitute for the fresh article when this cannot be obtained. Great care must be exercised during bad weather, especially during the extreme heat of the Red Sea, and suitable clothing must be provided and kept in readiness to meet the somewhat sudden transition from tropical heat to the piercing cold of the Mediterranean and Atlantic in winter.

As may readily be imagined, a chronic disease like sprue has been a fruitful field for the quack. His failures, of course, are frequent, but there can be little doubt that many cases of this disease have been cured by what I may call unorthodox methods. Van der Burg mentions a number of drugs and nostrums used in Java by a class of charlatans who profess to be "sprue doctors": in China, also, there are such people; one, in particular, acquired a great reputation in Shanghai and was extensively patronised. His "cure" consisted in dosing with castor oil for several days in succession—perhaps for a week—feeding the patient the while on milk. He then administered an aromatic tincture—possibly of simaruba with opium—along with about a dessertspoonful of a white powder containing a large proportion of carbonate of lime—possibly cuttle-fish bone or crabs' eyes finely powdered. On alternate days with the tincture and the powder he gave more castor oil. Generally in a week or ten days he allowed his patients beef-steak and potatoes in small quantity, and then gradually permitted them to return to ordinary diet. I have personal knowledge of at least two long-standing and severe cases of sprue who declare themselves cured by this man, and who now say they are quite well and able to eat and drink like other people. This man said the object of his treatment was to remove by means of the castor oil and massive powder the slime which, as he very correctly said, coats the gut.

PATRICK MANSON.

REFERENCES

1. BEGG, Dr. *Edinburgh Med. Journ.* Sept. 1890.—2. BERTRAND and FONTAN, Drs. *De l'entero-colite chronique endémique des pays chauds*, 1887.—3. *Brit. Med. Journ.*—4. BURG, Dr. C. L. VAN DER. *Chinese Customs Medical Reports*, 1, s. 27, 1888.—5. CHISHOLM. *Diseases of Tropical Countries*, 1822.—6. CUNNINGHAM, D. D. *Fourteenth Ann. Rep. of the San. Com. with the Gov. in India*, 1877.—7. DONALD. *Brit. Med. Jour.* 1st April 1881.—8. FAYRER, Sir J. *Tropical Diseases*, 1881.—9. HILLARY. *Observations on Diseases of Barbados*, 1756.—10. KELSCH and KIENER. *Maladies des pays chauds*, 1889.—11. LATHAM. *Med. Trans. Col. of Phys. Lond.* v. 1815.—12. MANSON, P. *Chinese Customs Medical Reports*, 1, s. 19, 1880.—13. THIN, Dr. *Practitioner*, Sept. 1883; ditto, 1887; *Brit. Med. Jour.* 14th June 1890; *Trans. Path. Soc.* 1892.—14. THIN and WETHERED, Drs. *Brit. Med. Journ.* 14th June 1890.

P. M.

INTESTINAL OBSTRUCTION

General observations.—Under the title of "intestinal obstruction" are included a great variety of conditions which, although unlike in character, have yet the common property of bringing about, mechanically, an obstruction to the passage of matter along the intestine. The obstructing agent may be, on the one hand, a peritoneal adhesion by means of which a loop of bowel is snared and acutely strangulated; on the other hand, it may be represented by a ring-like growth of carcinoma in the wall of the gut which very gradually narrows and perhaps closes its lumen. The bowel may be obstructed at one time by the torsion of a loop of gut around an axis at right angles to the line of its own course, as seen in volvulus of the sigmoid flexure; at another by the invagination of a certain portion of the bowel into the segment below it, as illustrated by intussusception. Again, the intestinal canal may be blocked by a gall-stone within its walls or be occluded by the pressure of a tumour entirely without its confines.

The symptoms produced by the various mechanical causes may be exceedingly acute on the one hand, or exceedingly chronic on the other.

The case may run its entire course from its onset to the patient's death in the short space of forty-eight hours, or the phenomena of obstruction may persist for years and even at the end may not be the direct cause of death.

When the clinical phenomena come to be reviewed it is at once evident that too much prominence must not be given to the mere fact that the bowel is obstructed. Obstruction of the bowel is the prominent symptom, but it is not the sole basis upon which the great issues of the malady depend.

In acute intussusception the disease
death without complete obstructio

the duration of the arrest of the passage of fæces an inevitable criterion of the gravity of the case. A loop of gut may be strangulated within the abdomen and death may follow within a week, no material having passed through the alimentary canal in the meanwhile; on the other hand, the colon may be plugged with a hardened mass of fæcal matter, no trace of a motion may be passed for four weeks, and yet the patient may make a good recovery. In the acute cases, as will be immediately shown, the mere fact that the bowel is obstructed is a comparatively unimportant one. The obstruction, as obstruction, does not produce the more urgent symptoms, nor does it act as the direct, nor, indeed, as the efficient cause of death. As an abstract proposition it may be stated that obstruction of the lumen of the bowel for a period of a week, or even more, is not in itself—all other circumstances being disregarded—a condition which need cause death or even distressing inconvenience.

If the clinical features of very acute and very chronic forms of intestinal obstruction be studied comparatively, the following general features become evident:—

In the very acute cases, as illustrated in strangulation by a band, the grave initial symptoms obviously do not in any way depend upon the undoubted fact that the bowel is obstructed. Time must elapse before the mere obstruction can produce definite phenomena of discomfort.

When a loop of gut is snared by a band, the initial symptoms are due solely to the sudden and severe injury to the peritoneum and to the numerous important nerves of the implicated part.

The symptoms, which are mainly those of intense abdominal pain, with collapse and usually with vomiting, have been described under the title of "peritonism" [see p. 616, article "Peritonitis"]; they are by no means peculiar to intestinal obstruction, but are common to nearly all acute lesions within the abdomen. Such symptoms may attend the passing of a gall-stone or the twisting of the pedicle of an ovarian tumour; and, indeed, both these conditions, during initial stages, have been mistaken for acute intestinal obstruction. The symptoms which depend upon the injury to the bowel—in distinction to the mere obstruction of its lumen—remain prominent for some little time, and, indeed, other things being equal, they depend in their degree of severity upon the amount of intestine involved, the tightness of the strangulation, and the nearness of the loop to the stomach.

Certain of the phenomena which follow depend undoubtedly upon the obstruction itself, and prominent among them must be placed the incessant vomiting and the distension of the belly. This latter symptom, however, is not solely dependent upon mere accumulation of matter in the gut above the narrowed part, as will be explained presently. Again, the final symptoms of acute intestinal obstruction—the symptoms which precede death—are not so much those due directly to occlusion of the lumen of the bowel, as those due to septic infection of the whole body from the disordered intestine.

The subjects of acute intestinal obstruction die for the most part with

the phenomena of septic poisoning, and if a certain stage of deterioration has been passed the mere relief of the obstruction does not save life.

In chronic intestinal obstruction, as illustrated by the slow closure of the colon by a ring of malignant growth, the phenomena are much more distinctly the direct outcome of the obstruction itself. The bowel fills up, but, so long as the obstruction is not complete and a little matter can escape from time to time, the distress occasioned may be slight. Two results follow. The irritation, septic and mechanical of the long-retained fæces leads to catarrh, the expression of which takes the form of a spurious diarrhœa. On the other hand, the bowel becomes hypertrophied in its persistent attempts to empty itself and coils in movement within the abdomen are usually to be seen and felt. A certain degree of septic intoxication is not uncommon even in fairly early stages of the disease, and the end is often by acute obstruction—as illustrated by the sudden blocking of the narrowed bowel, or by the septic poisoning which follows peritonitis. The infection, in those forms of peritonitis which depend upon intestinal obstruction, comes from the bowel, and in the surgical treatment of obstruction we have not only to relieve the mechanical cause of the trouble (as by dividing a band), but also to avert infection by emptying the engorged bowel of its putrid contents.

Varieties of intestinal obstruction.—The classification of intestinal obstruction is most conveniently founded upon the basis of morbid anatomy, and may be tabulated as follows:—i. Strangulation by bands and through apertures, including the so-called "internal hernias"; ii. Volvulus; iii. Intussusception; iv. Stricture; v. Obstruction by tumours and foreign substances within the bowel; vi. Obstruction by pressure of tumours, etc., external to the bowel; vii. Fæcal accumulation.

Relative frequency of the various forms.—Among 1000 fatal cases of intestinal obstruction, the above-mentioned varieties are roughly distributed as follows:—Intussusception, 350; strangulation by bands and through apertures, 250; stricture, 150; obstruction by tumours and foreign bodies within the bowel, 100; fæcal accumulation, 60; volvulus, 50; obstruction by means of tumours, etc., external to the bowel, 40: total, 1000.

This order of frequency is somewhat misleading, inasmuch as it is based upon such cases only as end fatally. In practice the cases due to fæcal accumulation, or reputed to be due to it, are the most common, and those due to foreign bodies and to the pressure of tumours and other diseases external to the gut are of greater relative frequency than the above list would appear to show.

An examination of the records of the London Hospital shows that the cases ascribed to fæcal accumulation are the most numerous; then come cases of stricture of the large intestine, then intussusception, and next in order of frequency strangulation by bands. Obstruction due to tumours external to the bowel rank next, and other forms may be spoken of as rare.

Portion of bowel involved.—Strangulation by bands is most commonly found in the small intestine. The small intestine also is most often concerned in internal hernias. Volvulus is most frequent in the sigmoid flexure, and, indeed, is rare elsewhere. Intussusception has its most usual seat in the ileo-cæcal segment of the bowel. Strictures of all kinds are more common in the colon than in the lesser intestine; and they are more common in the lower segments of the colon than in the upper. Obstruction due to pressure from without may be met with in any part of the canal, but more usually it concerns the large intestine as being the less movable; and especially the rectum and sigmoid flexure from their position with reference to the pelvis. Foreign bodies often lodge in the lower ileum and in the cæcum, and gall-stones are apt to become impacted in the jejunum or upper ileum. Fæcal accumulation can only occur in the colon, its more usual sites are in the cæcum and sigmoid flexure, or in the hepatic or splenic flexures.

Sex and Age.—*Strangulation by bands and through apertures* is a little more common in males than in females, is met with mostly in young adults, and is rare after forty. It is still rarer before ten. Internal hernias have been shown to be more common in males, and the greater number of the recorded cases have fallen between the ages of twenty-five and forty-five. *Volvulus of the sigmoid flexure* is about four times more common in men than in women. It is most common between forty and sixty, and, indeed, is rare before forty. *Acute intussusception* is a little more common in the male sex. It mostly attacks the young; 50 per cent of the cases are under the age of ten years. *Strictures* are equally common in the two sexes. The non-malignant occur about early middle life; the cancerous stricture is rare before forty. Certain strictures of the intestine are congenital. *Obstruction due to tumours external to the bowel* is obviously more common in women, and in adults of this sex. *Obstruction due to impacted gall-stones* is more common in women than in men, and the average age falls between fifty and sixty-five. *Fæcal accumulation* occurs with greater frequency in women than in men. It is most common in adults and in the aged, and is, as may be supposed, not infrequent in the insane and hysterical.

MORBID ANATOMY OF INTESTINAL OBSTRUCTION.—I. **Strangulation by bands or through apertures.**—Under this heading a great many different anatomical forms of intestinal obstruction are included. They all, however, have this common character—a loop of intestine, which may vary in length from a mere knuckle to many feet, is ensnared beneath an unyielding band, or compressed by the margin of some normal or abnormal aperture, and is thereby strangulated. The nature and effects of the lesion are precisely comparable to those which occur in strangulated hernia. The symptoms produced are acute, and, indeed, "acute intestinal obstruction" is best illustrated by this particular variety of strangulation. It follows upon this that the mortality from this form of obstruction is very high. Internal hernias are considered

under this heading. The symptoms which may attend this sub-variety are not, however, of necessity acute; they are acute when the involved bowel becomes strangulated, but if the accident be short of strangulation its manifestations may be subacute or even chronic.

The obstructing agents may be arranged in six divisions.

(A.) Strangulation by false ligaments. (B.) Strangulation by omental cords. (C.) Strangulation by Meckel's diverticulum. (D.) Strangulation by normal structures abnormally attached. (E.) Strangulation through slits and apertures, including internal hernias. (F.) Anomalous forms of obstruction due to isolated bands and to adhesions.

A. The bowel is strangulated by a peritoneal false ligament.—These so-called ligaments are isolated adhesions, the result of peritonitis. The peritonitis in question is commonly that of perityphlitis, or it may have depended upon pelvic peritonitis, or upon injury or strangulated hernia, or it may have been of tuberculous origin. Adhesions capable of producing strangulation may, indeed, follow any form of peritonitis from which recovery has occurred. Most of the adhesions formed during the course of peritonitis no doubt disappear.

During the progress of peritonitis the intestines are relatively still and are more or less distended from paralysis of their walls. As a result of this distension coils of bowel may be brought together that were hitherto far apart, or a certain loop may be placed in association with a comparatively distant point on the parietes. When the inflammation has subsided the parts return as far as possible to the normal state, peristaltic movements spread through the intestine, coils that were close together tend, as a result of those movements, to become separated, and adhesions which attach the intestine to points upon the parietes are persistently dragged upon. It follows from this almost constant tension that the still soft adhesion of recent peritonitis yields, becomes elongated and thinned, ultimately gives way, and is absorbed.

Movement also has great influence on the future physical condition of the adhesion. Most of the adhesions assume primarily a membranous character, and this they may retain throughout their existence. It is not uncommon to find some coils of intestine matted together by an extensive series of false membranes which appear sometimes as wide expansions at other times as thin but broad ribbon-like bands, of all dimensions and of various lengths. If two distinct coils of small intestine have been brought together during peritonitis, and have become attached to one another by means of the exudation, or if a like attachment has taken place between the intestine and the parietes, then, as movement is restored in the bowel, the adhesions, which may be quite membranous, are dragged upon, and as a result become elongated. As they increase in length so must they become attenuated in width and thickness. The constant tension, moreover, probably interferes with their already feeble nutrition, and induces some positive wasting. Thus the wide membranous adhesion may become narrowed and ribbon-like.

It may, however, undergo a still further change. This adhesion

subjected to the rolling movements of the intestines over one another, and subjected to frequent torsion now in one direction and now in another, tends to become rounded and cord-like, and the more it is stretched the more completely is this transformation favoured. Thus are formed "peritoneal false ligaments" and the bands which are so common a cause of strangulation of the bowel.

The moulding of the mass of adhesion-tissue into a cord by the movements active within the abdomen is illustrated by the changes that are effected by those movements in the omentum when it becomes adherent. This structure may become attached by its free extremity, and in the course of time, if the abdomen were opened, it would be found changed into a cord-like mass. The intestines in their movements have rolled over and under and about the adherent membrane until at last they have moulded it almost as a piece of clay may be moulded when rubbed between the palms. This change is best brought about when the situation of the adhesion is such as to keep the membrane on the stretch. A like metamorphosis may be effected in any smaller part of the great omentum that may have become adherent to a distant point.

By a combination of these various conditions, by a stretching of the adhesion on the one part, by its consequent attenuation on another, and its subjection to the moulding influences of moving intestines on the third part, it happens as a result of peritonitis that cords and bands of great length are often produced. Many instances may be given, but one of the most striking is afforded by a case reported by Mr. Obre, in which a cord-like band was found to pass from a coil of small intestine situated near the xiphoid cartilage to the parietal peritoneum about the inguinal canal. The false ligament measured 17½ inches. The patient had had a strangulated inguinal hernia, and there was clear evidence to show that the herniated bowel had been that to which the cord was attached.

It must be remembered that not only may these bands form arcades beneath which coils of intestine may become strangulated, but the longer of them may become separated at one of their points of attachment, and so form floating cords that may lead to strangulation of a loop by "knotting."

Some of the adhesions formed after inflammation of the serous membrane are exceedingly complicated, while others are found to unite parts usually remote from one another. Thus I find instances of adhesion between the ascending colon and the ovary, between the transverse colon and the cæcum in one case, and in another between it and the mesentery over the lower lumbar region, and again between the arch of the colon and a part of the parietes not far above the symphysis. Then again the sigmoid flexure has been found connected by adhesions to the bladder, the uterus, the rectum, the peritoneum in the right iliac fossa, and the cæcum. In all these cases I think that the unusual connection of parts may be explained by displacement from distension, the distension occurring during the progress of the peritonitis. Thus the transverse colon when

distended is apt to become bent upon itself, and by such bending to reach the lower parts of the abdomen. In the same way the distended sigmoid flexure may turn down into the pelvis, or extend across to the right iliac region, or even mount up in the abdomen and reach the liver.

Most commonly the "band" takes the form of a firm fibrous cord about the size of a No. 4 or No. 6 catheter, or it may be still more slender, and appear as a tough, rigid thread. On the other hand, it may be of comparatively large size; thus, M. Terrier has reported a case of internal strangulation where the constricting band had nearly the dimensions of the little finger. Less frequently the constricting agent has the appearance of an actual band, and in such cases is found as a tough, ribbon-like membrane, with a width of half an inch or even more.

A single false ligament, the representative of a single adhesion, may be produced by the very localised peritonitis that is sometimes associated with caseous degeneration of a mesenteric gland. A single adhesion may readily follow upon the little speck of peritonitis that often attends an intestinal ulcer.

However, a great many of the cases of "solitary band" described in records of cases are evidently instances of strangulation by Meckel's diverticulum, or by a diverticular ligament.

In some few cases two or more false ligaments have been found in the abdominal cavity. Sometimes these would appear to have been produced by the thrusting of a coil of intestine through a broad peritoneal adhesion so as to divide it into two segments; in another instance the bands are independent of one another. Mr. Berkeley Hill reported a case of acute intestinal obstruction where two bands existed, both of which constricted knuckles of small intestine. One constriction was comparatively slight, the other was severe; laparotomy was performed, and, unfortunately, the band found and divided was that associated with the minor obstruction.

Most commonly the strangulating band is connected by one end with the mesentery. In one very frequent variety the band is attached by both its extremities to the mesentery, the points of attachment being at a variable distance apart. In that very large series of cases where the isolated adhesion is due to pelvic peritonitis, it may be found attached by one end to some pelvic viscus, and by the other to a neighbouring part. Thus bands are found passing from the uterus, or ovary, or bladder to the parietal peritoneum of the pelvis or abdomen; or, starting from the same source, they may attach themselves to the cæcum or sigmoid flexure, or with much greater frequency to some part of the lower ileum or its mesentery. Other positions of attachment have been described already.

Modes of strangulation.—When a portion of the intestine is strangulated by an isolated peritoneal adhe on the gut will be found con-
stricted in one of two ways: (*a*)
band as beneath a shallow and
and constricted by a noose or loo

(*a*) Strangulation beneath a band can only occur when the band is comparatively short, and when it is stretched along a firm surface. From an examination of cases where the constricting cord is well described, it would appear that its average length in this form of strangulation is about 1½ to 2 inches. The arch beneath which the implicated bowel passes is variously described as large enough to admit one, two, or three fingers. As the band must be stretched along a firm surface it happens that this form of strangulation is much more commonly found about the posterior abdominal parietes than elsewhere. It is often met with about the iliac fossæ, especially that of the right side, and about the brim of the true pelvis. When a band passes between two points on the mesentery a coil of small intestine may readily be strangulated beneath it, the resisting parts between which the bowel is compressed being the false ligament on the one hand and the mesentery on the other.

(*b*) Strangulation by a noose or knot requires a long false ligament which must lie loose and free in the abdominal cavity, being attached only by its two ends.

The snaring of a coil of small intestine by this means must be a rare event, and in cases where the bowel is perfectly normal must be almost impossible. As Leichtenstern has well pointed out, it will usually be found in these cases that the gut was in an abnormal condition before the occurrence of the strangulation. A knuckle of gut may be rendered so adherent that when it had become involved in the noose or knot, it could not slip out of the way. It is probably still more common for two ends of a loop of intestine to be matted together by a little mesenteric peritonitis, so that if a noose should slip over such a loop, the constricting cord will find at the base of the loop a narrowed neck around which it may take good hold. The most common way in which a coil of intestine may be snared is when the lax band forms a ring or spiral between its fixed points. Strangulation by the formation of a knot is somewhat different from the process of snaring just described. The results are the same, and as the matter has no special practical bearing it need not be detailed in this work.

B. **Omental cords** may cause strangulation.—A portion of the free border of the great omentum may become adherent at some spot (commonly a hernial orifice, or near to a once-inflamed vermiform appendix), and the part so held may become changed into a cord-like structure. Sometimes the adherent part of the omentum is quite separated from the rest of the epiploon, or the whole mass of the omentum may be found rolled up into a fan-shaped structure, the base of the fan being at the transverse colon.

The mode of formation of these omental cords has already been described (p. 798).

One of the most curious modes of forming omental bands was met with in a case described by Dr. J. K. Fowler. Here the epiploon was divided into two lateral cords which, coming off from each side of the transverse

colon, passed down behind or among the intestines, and were found united together near the pelvis. All the patient's troubles dated from a kick received upon the abdomen. In this case it is probable that a rent had formed in the omentum, through which the great bulk of the small intestines had protruded. The lateral parts of the omentum—that is, the parts on each side of the rent—had then shrunken into cord-like masses which would be more or less hidden by the bowels.

As a rule the omental cords are much coarser and thicker than are the bands resulting from peritoneal adhesions. Many are nearly as thick as the finger, while a few only are described as very fine. In the matter of length they usually have an advantage over the simple band, as may be expected from the dimensions and relations of the great omentum.

The modes of strangulation by omental cords are identical with those described in connection with peritoneal bands, although it would appear that the proportion of cases of strangulation by a noose or knot is greater in the former than in the latter class of adhesion. This circumstance is no doubt due to the greater average length and the greater mobility of the omental false ligament.

C. **Strangulation by Meckel's diverticulum.**—The true or Meckel's diverticulum is due to the persistence or incomplete obliteration of the vitelline duct. When met with in its most perfect state it exists as a tube, having a structure similar to that of the small intestine itself, that extends between the lower part of the ileum and the umbilicus. The abdominal end of the tube opens into the lumen of the lesser bowel, while the umbilical extremity may be closed, or may open upon the surface and permit the discharge of fæcal matter. This condition, however, of the diverticle is comparatively rare; most commonly it exists as a blind tube coming off from the ileum. The length of this tube is on an average three inches, and in the great majority of the examples the measurement extends between one inch and four. Sometimes it exists only as a nipple-like projection. On the other hand, cases are recorded where the diverticle, in the form of a free tube, attained the length of ten inches. As a rule the abnormal tube is cylindrical in shape, with a conical extremity. In nearly every instance the intestinal end of the diverticulum is larger than its opposite end. In diameter its base is usually less than that of the gut from which it arises, although sometimes the diameters of the two tubes may be nearly identical. It may retain the same width throughout, and thus resemble a glove-finger. Much more frequently, however, its free extremity is considerably narrower than its base.

In structure the diverticle is composed of all the layers of normal small intestine. The muscular coat is sometimes deficient at the apex of the diverticle, and at this spot, therefore, hernial protrusions of the mucous membrane under the serous coat are not infrequently met with. When this occurs the extremity of the abnormal tube presents an ampulla of globular shape, and the process is said to be "clubbed." The

clubbed extremity of the diverticle, when it exists, takes an important part in the production of strangulation by knotting.

The diverticle is always single, and arises from the ileum from one to three feet above the ileo-cæcal valve. It is extremely rare for the process to take origin beyond these limits.

The process may come off at an acute angle with the long axis of the bowel, but more usually the angle formed is a right angle.

The end of the diverticle, in the majority of cases, is free. Very often, however, it is continued in the form of a solid cord. This cord should be attached to the umbilicus or to the abdominal parietes immediately below that cicatrix, an attachment, indeed, very frequently met with. Often the cord is pervious for a little way, and presents a minute canal into which a bristle may be inserted.

This diverticular ligament may break from its attachment to the parietes and may float free within the abdominal cavity. Under such circumstances, however, it is much more usual for it to acquire fresh adhesions to some point of the peritoneal surface. These secondary adhesions of a free diverticle, or of a diverticular cord at the extremity of one of the processes, are of considerable importance in the etiology of strangulation of the intestine. It is by the acquisition of a fresh point of attachment by the diverticle that constriction of the bowel is most often effected. In the great majority of cases the tube or the cord continued from it is adherent to the mesentery.

In another series of cases the diverticle does not exist as such, but is replaced in its entire length by a fibrous cord, identical in aspect with the band so often seen attached to the apex of the tubular process. These cords may be found to extend between the parietes in the vicinity of the umbilicus and that part of the ileum from which the more familiar diverticle takes origin. They may be considered to represent an entirely obliterated diverticle, or may be the remains of persisting omphalo-mesenteric vessels.

These diverticular ligaments may break loose from their connections at the umbilicus, and, like the tubular processes, may either remain free in the abdominal cavity, or form secondary adhesions at almost any spot.

To complicate this matter still further, the cord may retain its attachment to the anterior abdominal wall, and then separate from its connection with the intestine, either forming no other attachment, or becoming adherent to a point somewhere within the abdomen.

Finally, a cord may stretch from the root of the mesentery to be attached to the margin of the ileum (close to its mesentery), opposite the spot from which the diverticle most commonly arises. Leichtenstern believes that such bands represent that part of the omphalo-mesenteric vessels which extends between the bowel and the main blood-vessels at the root of the mesentery.

There can be little doubt that these strangely attached diverticular ligaments have often been mistaken for isolated peritoneal adhesions;

and, in any case, where a "solitary band" exists without a trace of ancient peritonitis, there are at first sight some grounds for suspecting the cord to be of congenital origin.

Modes of strangulation.—A coil of small intestine may be strangulated beneath an adherent diverticulum precisely in the same manner as it would be when beneath a peritoneal adhesion. A diverticular ligament, whether attached to the extremity of a pouch-like process, or (in the absence of such process) connected directly with the gut, may form precisely the same kinds of noose and knot as are formed by isolated adhesions. The length and looseness of the congenital ligament render it well able to snare the bowel, provided that the position and circumstances of the bowel render it apt to be snared.

Of this character, probably, are the numerous specimens found in museums where these cords are seen to have made one and a half or two turns round the involved bowel.

Strangulation by knots formed by a free diverticulum.—These remarkable knots and the methods of their formation have been very exhaustively studied by M. Parise. To produce these knots it is necessary that the diverticle should be of good length, should be quite free (save only for its intestinal attachment), and should possess an ampulla at its extremity. The importance of the ampulla is paramount, and French writers are in the habit of speaking of it as *la clef de l'étranglement.* Three varieties of knot have been described. The details of these complex forms of strangulation need not be entered into. I have described them in my work on *Intestinal Obstruction.*

Strangulation over a diverticular band.—In this form a loop of intestine is thrown over a tightly-drawn diverticular band as a shawl is thrown over the arm. Under certain conditions, which need not be detailed here, an obstruction follows in the bowel so displaced. The occlusion is somewhat similar to that which would take place in a coil of thin india-rubber tubing, if thrown across a tense wire cord and allowed to become dependent (see p. 808).

Strangulation by kinking.—If, under certain circumstances, much traction be brought to bear on a diverticular ligament, the gut, without undergoing any structural alteration, may become so acutely bent at the point of origin of the abnormal band or process as to be occluded. It has been shown also that a free diverticle, when of good size, and coming off at about a right angle with the bowel, may cause such a bending of the bowel when the pouch is so much distended as to cause obstruction (see p. 809).

D. **Strangulation by normal structures abnormally attached.**—*The vermiform appendix* may become adherent to some point on the neighbouring peritoneum, and so form a band or arch beneath which intestine may be strangulated. The process is very commonly adherent to the mesentery of the lower ileum. Less frequently it is adherent to the ileum itself, or to the cæcum, or to the peritoneum about the right iliac fossa and margin of the pelvis. In one instance the appendix was

adherent to an enlarged ovary on the right side, and beneath the cord so formed a loop of the ileum and a part of the ascending colon were constricted.

In some rare cases the appendix has been described as wound in the form of a close spiral, or of a ring into which a loop of intestine has entered and has become strangulated. In other instances, equally uncommon, the appendix is said to have tied itself into an actual knot of a character similar to those sometimes formed by the true diverticulum. By such a knot the bowel has been constricted.

In several instances *the Fallopian tube* has become adherent to some part of the neighbouring peritoneum, to that, for example, lining one of the iliac fossæ; and beneath the arcade so formed a portion of the small intestine has been strangulated.

A few cases are reported where a loop of bowel has been strangulated beneath a band formed by *a fixed portion of the mesentery.* In these examples some coils of the small intestine have become fixed at a distant spot. They may be involved in a large irreducible hernia, or may have hung down into the pelvis, and acquired adhesions when in that position. Under such circumstances the corresponding part of the mesentery may become tightly stretched across the posterior wall of the abdomen or the pelvic brim, and a bridge be thus formed, beneath which some of the lesser bowel may become strangulated.

Instances have also been reported in which bowel has been found strangulated by bands formed by adherent appendices epiploicæ, and in other examples by the pedicle of an ovarian tumour.

E. **Strangulation through slits and apertures, including internal hernias.**—Through holes formed in the mesentery portions of intestine have frequently been strangulated. The holes are usually slit-like, and are most common in the mesentery of the lower ileum. In many cases these slits can be more or less distinctly traced to an injury; in other cases there is every reason to believe that the abnormal aperture is congenital. The edges in these instances are smooth, rounded, and regular; there is no history of injury, and no trace of any previous peritonitis. I have given an account of these congenital holes in my Hunterian Lectures on the *Anatomy of the Intestinal Canal and Peritoneum in Man.* The hole is usually placed near to the intestine. It varies in size. It may be no larger than a sixpenny piece, or it may admit several fingers.

Mr. Partridge has recorded a case in which a knuckle of ileum was strangulated through a slit in the mesentery of the appendix.

Bowel may be snared in slits formed in the omentum. These slits may be of congenital origin, but many are undoubtedly due to injury.

Mr. Holmes records a remarkable case where a loop of the lower ileum was strangulated through a hole apparently formed in an appendix epiploica. The appendix in question belonged to the sigmoid flexure, and formed a fatty fibrous ring through which a loop had passed.

I have operated successfully upon a case in which repeated attacks of

strangulation were due to a snaring of the bowel in a slit in the sustentaculum lienis.

Bowel has been strangulated by the margins of a slit in the broad ligament, by a like gap in the suspensory ligament of the liver, and even by a slit in a broad membranous adhesion.

Internal hernias.—The following brief account is abstracted from my article on this subject in *The System of Surgery*, vol. ii. 1896. The symptoms, such as they are, are given in the account of each variety:—

Varieties.—The following varieties come under the above heading:—(*a*) Diaphragmatic hernia; (*b*) Hernia into the fossa duodeno-jejunalis; (*c*) Hernia into the foramen of Winslow; (*d*) Intersigmoid hernia; (*e*) Perityphlitic hernia.

(*a*) Diaphragmatic hernia.—In this lesion certain of the abdominal viscera, particularly the stomach, colon, and omentum, are thrust through a hole or rent in the diaphragm into the thorax. Less often the herniated organs are the liver, spleen, and small intestine. The aperture in the diaphragm may be due to injury or to congenital defect. The congenital defects are more common on the left side. [*Vide* art. "Diaphragmatic Hernia," p. 577.]

A diaphragmatic hernia may become strangulated.

Symptoms.—The general symptoms of diaphragmatic hernia are pain, dyspnœa, inability to lie on the affected side, displacement of the heart and lung, distension of one side of the chest, and abnormal hyper-resonance and sinking in of the belly. To these may be added the phenomena of intestinal obstruction.

The only treatment is by operation.

(*b*) Hernia into the fossa duodeno-jejunalis.—Pathology.—This hernia is known by many names, of which the following are the chief:—Retro-peritoneal hernia, mesocolic or mesenteric hernia, mesogastric hernia, duodenal hernia, and the hernia of Treitz. The fossa duodeno-jejunalis is formed by a fold of peritoneum at the point where the duodenum ends in the jejunum. Out of 100 bodies examined I found this fossa in 48. I have figured it in my article on the peritoneum in Morris's *Treatise on Anatomy*, p. 1002.

In this remarkable hernia the fossa in question becomes deeper, and accommodates more and more intestine. The upper jejunum enters first, and then the small intestine from above downwards. In a complete case, when the abdomen is opened, nothing of the alimentary canal save the stomach, duodenum, and colon are to be seen. The whole of the small intestine is lost to view in an enormous sac which is placed behind the posterior parietal peritoneum. The duodenum can be seen to enter the sac and the end of the ileum to leave it. The sac usually extends downwards on the left side, and may reach the promontory of the sacrum. It lies in the retro-peritoneal tissue. The cæcum and ascending colon have their normal positions, but the transverse and descending parts of the colon are stretched over and displaced by the sac. In its upper part the sac may reach to the spleen.

The orifice of the sac is usually to the left of the spine and near the ending of the duodenum. It may be displaced to the right iliac region.

The sac, as already stated, may contain the whole of the small intestine, except the duodenum. On the other hand, it may contain a few feet of the jejunum only. As a rule these hernias are large. Strangulation, when it occurs, is always brought about by the neck or orifice of the sac.

Symptoms.—About seventy examples of this hernia have been recorded. It is more common in men than in women in the proportion of three to one. It has been met with at all ages, but is most common between twenty-five and thirty-five.

In many of the cases the hernia was discovered post-mortem, and there was no evidence that it had given much if any trouble during life; in other instances the patients were the subjects of continual dyspepsia, constant colic with constipation and occasional vomiting, and much flatulent distension of the abdomen. In some a cyst-like swelling, resonant or partly resonant on percussion, was noticed to the left of the umbilicus. In a third series of cases the symptoms were those of chronic intestinal obstruction, with constipation, colic, and vomiting. A dull, soft, well-defined tumour was now and then noted in the left side of the abdomen. In the region of the right colon the abdomen was retracted. In a fourth series of cases there was acute strangulation, death ensuing in from two to eighteen days.

I am not aware that this hernia has ever been diagnosed during life, or that any operation performed upon a subject of it has been successful.

(*c*) Hernia into the foramen of Winslow.—The hernia takes place through the foramen of Winslow, the protruded viscera entering the lesser sac of the peritoneum behind the stomach. Eight cases at least have been recorded.

It is met with in adults, and in men more often than in women. The part of the bowel involved is usually a part of the colon. It is rare for any large part of the small intestine to enter into the hernia. In some instances the rupture was discovered post-mortem, and nothing was known of the clinical history. In most of the other cases the symptoms were those of acute strangulation. I have reported a case in a man aged twenty-six. The symptoms were acute. I performed laparotomy and discovered the cæcum, ascending colon, and part of the ileum strangulated by the margins of the foramen. All attempts at reduction failed, and at the autopsy I was unable to reduce the bowel until I had divided the hepatic artery, portal vein and bile-duct.

(*d*) Intersigmoid hernia.—A fossa or pouch of peritoneum is often found in the sigmoid mesocolon. It is formed by the layers of the mesocolon, and is produced by the sigmoid artery. It is placed over the bifurcation of the iliac vessels, and is known as the intersigmoid fossa. It is funnel-shaped, and opens to the left. A hernia may take place at this spot.

The sac formed by the gradual distension of the pouch may be small;

but in one case it was as large as an adult head, and contained two-thirds of the small intestine. Three cases have been recorded, two in women and one in a man; the ages were respectively fifty-seven, sixty-three, and sixty-five. In two cases the symptoms were acute and rapidly fatal. In the other case there were chronic bowel troubles, colic, vomiting, constipation, thirst, frequent micturition, and pain and swelling in the left iliac fossa. This case ended acutely.

Treatment by laparotomy is the only measure available.

(*e*) Pericæcal hernia.—Certain fossæ are met with about the cæcum; they are formed from the peritoneum, and the two chief are the superior and the inferior ileo-cæcal.

Hernias have occurred in one or other of these pouches.

The sac is usually small, and passes upwards behind the cæcum and ascending colon. Usually it has contained coils of the lower ileum.

Some twelve examples have been recorded. In four the hernia was discovered after death, and the bowel found free from any strangulation. In the other eight cases the patients died with symptoms of more or less acute strangulation in the course of a few days. In most of these instances the onset was sudden; in a few there had been long-existing intestinal uneasiness. The proportion of males to females was as four to one, and the ages ranged from twenty-five to forty-five.

An early laparotomy represents the only measure of treatment.

F. Anomalous forms of obstruction due to isolated bands and to adhesions.—Under this heading may be grouped a series of cases, all more or less infrequent, in which an obstruction has been brought about by means of an adherent diverticulum, or by an isolated band, or by more extensive adhesions, but where the mechanism of the occlusion is unlike that involved in the cases already described.

These cases are united by a common pathological bond, though clinically they present conspicuous differences.

It will be convenient, as the examples are exceptional, to discuss the symptoms and the pathology together.

These anomalous cases may be classified under the following headings:—

(*a*) Strangulation over a band.
(*b*) Occlusion brought about by acute kinking due to traction upon an isolated band or an adherent diverticulum.
(*c*) Occlusion effected by adhesions which retain the bowel in a bent position.
(*d*) Obstruction by means of adhesions which compress the gut.
(*e*) Obstruction by the matting together of several coils of intestine.
(*f*) Narrowing of the bowel from shrinking of the mesentery after inflammation.

(*a*) Strangulation over a band.—If several coils of a thin india-rubber pipe, through which water was flowing, were thrown over a tightly-drawn wire, the lumen of the tube would become more or less completely

occluded at the spots of crossing the wire. It is conceivable that a similar consequence may arise in the abdomen when a long loop of intestine is thrown across a more or less rigid band. Here the weight of the dependent loop would act as a compressing agent, and the interference with the circulation in the mesenteric vessels would induce an engorgement of the involved bowel and some meteorism. It is difficult, however, to understand how such a form of obstruction could occur in the living subject without some arrangement of parts which would permit the dependent coils to retain their position. In two recorded instances an extensive loop of the lower ileum had passed through a large hole in the omentum; the loops were black with congestion, and were hanging down into the pelvis. In one case the coil was fixed in this position by recent adhesions. In neither of the cases was the obstruction effected by the aperture itself, the gut being very readily withdrawn at the autopsy. As the recorder of one of the cases (Dr. Fagge) observes, the strangulation was not due to the narrowness of the aperture, but to the hanging of the gut over its lower edge. In both cases the symptoms appeared suddenly; in both acute peritonitis was found at the post-mortem; in both the patient lived five days. In another case a diverticulum passed to be attached to the umbilicus, and over it two loops of the ileum, black with congestion, were suspended. They were found twisted upon themselves, and it is impossible to say which was the primary and more essential phenomenon, the volvulus or the hanging of the gut over the cord. The symptoms appeared suddenly, acute peritonitis set in on the sixth day, and the patient died on the ninth.

The symptoms, taken generally, do not differ from those of acute intestinal obstruction, as illustrated by strangulation by a band.

(*b*) Occlusion by acute kinking due to traction.—In these cases a band attached to the bowel so drags upon its point of attachment that the gut becomes acutely bent at this spot, and is ultimately occluded by a process akin to the kinking that may close an india-rubber tube. This condition is usually met with in the case of a diverticulum or diverticular ligament attached to the umbilicus; or in instances where an isolated adhesion is connected with the ileum on the one hand and some more fixed and distant point on the other.

Dr. Reignier has shown that it is possible for an unattached diverticle to cause obstruction by kinking, if the process become much distended. In the body of an infant he found a free diverticle 7 centimetres long; on injecting water into the gut above the process, he found that when the pressure was moderate the diverticle simply became filled and that the fluid passed readily by it; when, however, the pressure was much increased the process dilated enormously, and so pressed upon the gut below its point of origin as to bend the intestine transversely and finally to occlude its lumen. He gives a case in the person of a man, aged twenty-two, which illustrates this experiment in practice. This patient died after exhibiting for ten days the symptoms of acute intestinal obstruction. The autopsy showed a free diverticulum,

much dilated by liquid fæces, which had bent the gut from which it arose so acutely that the lumen of the intestine was quite closed. On lifting the diverticle and gently pressing it the obstruction was at once overcome.

So far as can be judged from the few cases published, the symptoms due to kinking of the bowel are practically identical with those due to strangulation under a band.

(*c*) Occlusions by adhesions which retain the bowel in a bent position.—In these cases, which concern both the large and the small intestine, the gut is found to have become adherent to some fixed point in such a way that a more or less acute bend is produced. The site of the adhesion is on the abdominal or pelvic parietes or the pelvic viscera. It may be on the liver. The usual cause of the adhesion is either pelvic peritonitis or hernia.

In the case of rupture the part of the bowel adherent is the same that occupied the hernia. The condition is met with, therefore, only after enteroceles, and only after such as have been strangulated or inflamed. The bowel, presenting in any case some inflammation of its serous coat, is reduced into the abdomen, and, instead of remaining free in that cavity, contracts adhesions with some other part of the peritoneum. In every case of this kind, so far as I am aware, the adhesion of the bowel has been to the parietes in the vicinity of the hernial orifice.

The bowel, having been recently herniated, usually acquires an adhesion in a bent position, and when so fixed often leads to further intestinal troubles in cases where strangulated or inflamed hernias have been successfully reduced. The involved gut is usually adherent at one isolated spot only, and a single and simple angular bend is thus produced. This is the condition met with in those cases that depend on hernia. In other instances the attachment may be more extensive, as in a case of Dr. Fagge's, where one foot of the lower ileum was found adherent to the anterior abdominal parietes as a result of omental sarcoma. Moreover, the bends formed in the bowel may be by no means simple. There may be several angular bends, the loops being adherent at more points than one, and made to assume the outline of the letter N. This arrangement may be still further complicated by the matting together of the three bars of the intestinal N, whereby the false position is perpetuated. In one case where N-like bends were produced only 4 inches of bowel were involved, so that the angles formed were very acute and abrupt.

A few examples may be given to illustrate the varieties assumed by this form of intestinal obstruction. The convexity of the ascending colon may become adherent to the ovary, and the gut may be so narrowed at the bend as barely to admit a crow-quill. The transverse colon may become adherent to the fundus uteri. The rectum may attach itself to a cancerous ovary, and present in consequence a very angular bend. The sigmoid flexure may adhere to a uterus the seat of malignant disease and present so abrupt a bend that fatal obstruction with symptoms like those of a volvulus may ensue.

The interval that may intervene between the formation of the a

hesion and the occurrence of symptoms of intestinal obstruction varies greatly. Evidences of obstruction may appear within a few days of the original lesion, but in the great majority of cases the intestinal symptoms do not make their appearance until months after the initial peritonitis: sometimes years have elapsed between the causative inflammation and the symptoms of obstruction, such examples being most usual in the large intestine. Many of the patients have been the victims of chronic constipation for years before the final occlusion occurred. Adhesions of the same character as those now under consideration have been met with in the necropsies of patients who during life presented no marked intestinal symptoms.

The mechanism of the obstruction in these cases varies, and may be conveniently considered in three categories, taken in order of severity:—

(α) The gut at the adherent point may become so bent that occlusion by kinking is produced. The symptoms induced are severe and sudden in their onset.

(β) The bowel (a portion always of the small intestine) is adherent over a small area, and symptoms of obstruction follow from certain effects of traction without conspicuous occlusion of the lumen of the tube. It is certain that, so far as the lesser bowel is concerned, mere adhesion over a limited area tends to cause an impediment to the passage of matter. The gut at the adherent spot cannot exercise its peristaltic function; it becomes a more or less inert segment in an active tube. If a little acute mischief be excited about the seat of the adhesions, symptoms of an acute or subacute character may arise, the exact pathogenesis of which is obscure.

(γ) The adherent bowel may offer a more or less definite mechanical obstacle to the passage of its contents. A part of the colon may present so sharp and rigid a bend as to give to the involved intestine the properties of a stricture. In other instances the bowel, and particularly the lesser bowel, is adherent over a wide area, and the mere inertness of the attached portion constitutes an obstruction. This is well seen in those cases where the bowel is adherent in a contorted position, or when several inches of it are blended in a straight line with the parietes. Here the bowel above the diseased part has not only to pass its own contents along, but has to force them also through the inert and adherent segment. The longer this segment the more marked the obstruction. When closely bound down, the involved gut must be practically incapable of peristaltic movement.

The symptoms associated with this form of obstruction will obviously show great variation. They may assume an acute, or a subacute, or a chronic aspect, and may differ somewhat, as the occlusion is situate in the large or the small intestine. If the obstruction be due to a sudden closure of the gut by kinking at the already bent and adherent part, the symptoms may be of a very acute character.

This condition appears to occur most usually in connection with the sigmoid flexure or rectum, and the manifestations produced are identical

with volvulus of the former segment of the bowel, and cannot be distinguished from it.

The symptoms may be subacute, as in a case reported by M. Cour where the sigmoid flexure was adherent to a cancerous ovary. Here the final attack lasted some eight or nine days, and was marked by paroxysmal pain with visible peristalsis, by slight non-stercoraceous vomiting, and by constipation relieved by an occasional stool. In other instances the manifestations may be quite chronic, and may resemble in all points those due to stricture of the rectum.

When the small intestine is involved there may be acute symptoms from sudden kinking. In most of the examples there were evidences of incomplete obstruction; and constipation alternated with an occasional motion. In some of the less acute cases the patient, when not absolutely constipated, passed many scanty and very liquid stools. The vomiting is not severe at first, and occurs at long intervals. The abdominal pain is paroxysmal. There is a dragging pain about the part to which the gut is adherent. There is not much distension of the abdomen. In the chronic cases the movements of the intestinal coils are visible.

(*d*) Obstruction by means of adhesions which compress the gut.—Peritoneal adhesions, when favourably placed, may undergo considerable contraction. When placed upon the bowel these false membranes may by their shrinking so compress the intestine as seriously to narrow its lumen. Experience demonstrates a fact that might have been anticipated, namely, that this form of constriction is most usually met with about the most fixed segments of the intestine, that is to say, about the ascending and descending colon, and the hepatic and splenic flexures.

The extent to which the bowel is narrowed in these cases is often considerable. In some the affected colon would barely admit the tip of the little finger; in others it would only admit a crow-quill.

I have found but few examples of this form of obstruction in the small intestine. In every instance there has been some complication in the case. The affected bowel is always adherent to the parietes or to the pelvic viscera.

As regards the symptoms incident to this variety of obstruction, it can only be said that they more or less completely resemble those due to stricture of the bowel. In the case of the colon this assertion may be made without reservation; in the case of the small intestine the manifestations of the disease appear more rapidly than is usual in stricture, the permanent stenosis being complicated by the effects of angular bending.

(*e*) Obstruction by the matting together of intestinal coils.—The many cases that can be classed under this category present a protean aspect.

(*a*) The small intestine.—The coils of the lesser bowel may be matted together in many different ways. In one set of cases a small segment of the gut is so adherent as to form a permanent and unchanging loop. This loop may be open, that is, the gut may only be adherent at the extremities of the loop, or it may be closed, that is, the coil of gut involved i

adherent throughout. In another set of cases, many coils, involving often a considerable tract of the intestine, are matted together so as to form more or less complicated masses. In both instances the involved coils are usually quite free from adhesions to the parietes or to other viscera.

Many of the cases in which a permanent loop has been produced are the results of hernia; others depend upon an ulcer of the bowel which has nearly perforated, or upon mesenteric gland disease. The two limbs of the loop may communicate with one another—at the neck of the loop—by means of a fistulous opening.

The matting together of numerous coils is very often the outcome of tuberculous peritonitis, or of the peritonitis following operation, disease of the pelvic organs, or perityphlitis.

The adherent coils usually form a round mass which may be almost as distinct as a tumour, and which compares conspicuously with the uninvolved and normal bowel. The matted intestine may be adherent to the parietes also, or it may be quite free. Sometimes the matting is brought about by a multitude of isolated adhesions. In other cases the coils are enveloped in fine membranous adhesions, so that they may appear as if enclosed in a bag of tough tissue paper. Some of the coils in the mass may be of normal lumen, others may be dilated, and many may be compressed; they are commonly strangely distorted. When obstruction has been caused the bowel entering the mass will be dilated, while that leaving it will be more or less shrunken. The length of gut involved varies from a few inches to several feet.

The symptoms are in the main identical with those associated with stricture of the small intestine. The onset is gradual, the progress of the malady is irregular, severe periodic attacks are common, and an acute termination to the case is not unusual. Constipation is partial, and often alternates with a copious diarrhœa. The vomiting is usually slight, irregular in occurrence, and uncertain in duration. During an exacerbation of the symptoms, and especially during a final acute attack, it may become stercoraceous. The pain, such as it is, is paroxysmal, decreasing as the case advances. Early in the case there may be an attack of colicky pain not more frequently than once or twice a week. Towards its termination the paroxysms may come on at intervals of a few minutes. The patient usually emaciates, and the movements of the distended and hypertrophied coils of intestine above the obstruction are, as a rule, evident through the parietes. There is little or no distension of the abdomen unless an acute form of obstruction supervene, and even in such a case the meteorism is usually by no means excessive. In two or three instances the mass of adherent bowel has been detected, as an ill-defined tumour, through the abdominal parietes. The duration of the case may be reckoned in months rather than in weeks.

(β) The large intestine.—The colon being a more or less fixed part of the bowel, it follows that it is not susceptible of quite the same morbid conditions as have just been described in association with the lesser bowel.

As a result of distension, however, parts of the colon may become greatly elongated, and the abnormal coils thus formed may become matted together by adhesions.

I can find no case where the descending colon, the most fixed part of this bowel, is stated to have altered its position to any conspicuous extent as a result of distension. In one instance a dilated ascending colon appears to have become so curved that its convexity was adherent to the ovary. The sigmoid flexure when distended is apt to stretch towards the right iliac region, and then to mount up into the right hypochondriac region. The two limbs of the dilated loop may be found matted together, or the summit of the loop may be found adherent to the cæcum, to the peritoneum in the right iliac or hypochondriac regions, or even to the under surface of the liver. The transverse colon undergoes a peculiar and common change when much distended. Its central point tends to pass downwards towards the pelvis, so as to produce a V or U-shaped bend. The apex of the V or the bend of the U may become adherent to the mesentery, or to the peritoneum in the pelvis, or to a pelvic viscus such as the fundus of the uterus. One limb of the V may become adherent to the whole length of the ascending colon and so produce a "double-barrelled ascending colon," or the other limb may attach itself to the descending colon in a like fashion and produce a similar appearance on the left side. In many cases this deformity of the colon has been the result of chronic obstruction in the lower part of the bowel, such as a stricture of the sigmoid flexure or rectum, or a fæcal accumulation.

There are cases where one limb of the bent colic arch is found adherent to the ascending or descending colon for its entire length, but where no obstruction of any kind is found in the gut below the distorted segment. I am disposed to believe that such cases depend upon ulceration of the colon. The ulceration leads to peritonitis, distension and distortion of the transverse colon may follow, and then a part of the altered arch may become adherent to the inflamed serous coat of the ulcerated bowel.

No abdominal symptoms may be excited by these conditions of the colon, although there is usually some evidence of chronic constipation. The matting of the sigmoid flexure in the way described is very apt to lead to volvulus of that part; and in the case of the deformed and adherent colic arch more or less acute obstruction may supervene from occlusion by kinking.

On the other hand, the clinical phenomena may be those of stricture of the colon of a not too prominent grade.

(*f*) Narrowing of the bowel from shrinking of the mesentery after inflammation.—When the mesentery has been extensively inflamed it may subsequently undergo such marked contraction as greatly to narrow the bowel to which it is attached. In such cases the involved coils are often found bound down to the spine by the shortened mesentery and much shrunken. This is very often the result of mesenteric gland disease.

I might refer to four well-marked examples of this form of contraction, all in young patients.

The symptoms which arise in these cases are practically identical with those of stricture of the small intestine, or with those of matting together of many coils of the bowel. It would appear from cases that the evidences of obstruction may extend over years; for example, for four years in one case, and for two in another.

II. Volvulus.—Under the general name "volvulus" are included two distinct modes of obstruction. In one the bowel is so twisted about its mesenteric axis that it becomes occluded; in the other form two suitable coils of intestine are so intertwined or knotted together as also to cause an obstruction in their canals.

The subject may be most conveniently considered under the following heads:—(A) Volvulus of the sigmoid flexure. (B) Volvulus of the ascending colon and cæcum. (C) Volvulus of the small intestine.

A. *Volvulus of the sigmoid flexure.*—This part of the bowel may be occluded by either of the two methods just named: (*a*) It may be twisted upon its mesocolic axis; (*b*) it may be intertwined with a suitable coil of small intestine.

(*a*) The bowel is twisted about its mesocolic axis.—This is the most usual form of volvulus, and may, indeed, be said to be the only form that is at all common. If all the cases of volvulus of the intestine be considered collectively, it will be found that of the number about four-fifths are instances of twist of the sigmoid flexure about its mesocolic axis.

The arrangement of the gut necessary for the production of a volvulus is the following:—The loop must be of considerable length, and the mesocolon must be long and very narrow at its parietal attachment, so that the two ends of the loop are brought as close together as possible. The outline of such a loop is that of a capital omega.

This arrangement of the parts may be congenital, although such a circumstance must be uncommon since volvulus of the sigmoid flexure is extremely rare in the young. It may be brought about by peritoneal adhesions, especially by such as have been formed after great distension of the bowel. It may be readily produced by inflammatory conditions in the mesocolon leading to cicatricial contraction.

The commonest cause, however, without doubt is chronic constipation. In this condition the flexure is more or less constantly distended, its walls become partly paralysed by the distension, and, becoming filled with fæcal matter and flatus, it hangs down into the pelvis an inert heavy mass. So placed it must drag upon its mesocolon, and while the position, on the one hand, tends to elongate that membrane, it appears, on the other, to approximate the two ends of the loop. The deformity produced may possibly be rendered permanent by some local peritonitis.

When the loop is in this condition it is easy to understand that a twisting of it upon its axis may be brought about, and, if a heavy loop blocked with fæces is concerned, the position of the body may become a

factor in the causation of the twist: lastly, distension of the bowel alone has great influence both in producing and maintaining a volvulus.

According to Potain there are two kinds of twist: in one the superior part of the loop is carried from above downwards, and from behind forwards, in front of the lower half of the loop, so that the end of the descending colon is brought in contact with and in front of the commencement of the rectum ("type rectum en arrière"); in the second form the superior part of the coil is carried from above downwards, and from before backwards, behind the lower segment of the loop, so that the end of the descending colon is brought in contact with and behind the commencement of the rectum ("type rectum en avant"). Of these two varieties the former is by far the more common. The twist may extend through an arc of 180° to 360°, or the bowel may be twisted twice or even three times about its mesocolic axis. Since at the root of the flexure the two ends of the loop are nearly parallel to the mesenteric axis, it follows that when the latter is twisted the former also must be twisted upon their own axes.

When once formed the volvulus soon becomes permanent, the heavy and distended loop being without power to straighten itself. Its wall becomes engorged with blood, and meteorism follows this interference with the circulation. The more the bowel becomes distended the more fixed is the volvulus. If the gas be let out the bowel can become untwisted, but if the distension be allowed to recur the volvulus forms again.

At the necropsy in fatal cases the sigmoid flexure is found enormously distended; it seems to occupy the whole abdominal cavity. In cases of slight distension the loop reaches to the umbilicus. As it becomes more distended it tends to move towards the right hypochondriac region; it then lies in front of the stomach, and ultimately reaches the liver. In severe cases the diaphragm is much pressed upon, and may be pushed up to within 16 cm. ($6\frac{1}{4}$ inches) of the clavicle, or even up to the level of the third or fourth rib.

The twisted coil is congested more or less intensely. In colour it may present any depth between a dark red and black. Its walls are often much thickened by infiltration, and are softened and friable. The serous coat is very commonly found to exhibit a rent, or even several rents; these may be extensive and may involve the muscular coat also, whilst the mucous membrane escapes. The walls of the flexure may become gangrenous in patches. I find that perforation has occurred only twice in twenty cases I have collected. Peritonitis is very common; it appears early, and begins on the twisted loop.

(*b*) The bowel is intertwined with a suitable coil of small intestine.—In these cases the sigmoid flexure must have the anatomical arrangement described in the preceding paragraph, that is, it must form a long, free loop with a narrow pedicle. The loop of small intestine should possess also an unnatural mobility, and should have an unduly long and narrow mesenteric pedicle. In cases where two such coils

have become intertwined, it is found that the loop of the lesser bowel varies in length from 4 to 21 inches, while that of the sigmoid flexure measures from 12 to 40 inches (Leichtenstern). The usual mode of intertwining is as follows: The loop of small intestine falls in front of or across the pedicle of the sigmoid flexure; the flexure then winds itself around the axis formed by the lesser coil, it passes upwards in front of the loop of small intestine, and then moves backwards and downwards, so that its free end passes behind the pedicles of the two coils. In this way the abnormal sigmoid flexure forms a complete turn around the coil of lesser intestine. Both segments of the bowel become strangulated, but the occlusion will be most severe in the axial loop. Other methods of intertwining may occur. Strangulation is always severe.

The symptoms are those of acute strangulation of the bowel. Diarrhœa often marks the early stage of the attack. The course is very rapid. Out of twenty-one recorded cases only one patient lived until the sixth day, all the rest died within twenty-four or forty-eight hours.

B. *Volvulus of the ascending colon and cæcum.*—Volvulus occurring in this part of the intestine may assume a variety of aspects, and, in any case, is apt to adopt a very complicated arrangement.

It may be considered under three categories: (*a*) A twist of the ascending colon around its own axis; (*b*) Twists brought about by an abnormal loop formed by the ascending colon and cæcum, with a long and distinct mesocolon; (*c*) Twists of the cæcum "upon itself," or about its own axis.

(*a*) Occlusion of the bowel may be brought about by a twist of the ascending colon around its own vertical axis. It would appear that this condition may be found in a colon that presents no anatomical abnormalities. It is extremely rare.

(*b*) The cæcum and ascending colon, as a result of incomplete development, may be perfectly free in the abdomen, and be attached by an extensive mesocolon continuous with, and as extensive as, the mesentery.

I have given an account of this condition in my Hunterian Lectures on the *Anatomy of the Peritoneum and Intestinal Canal in Man.*

The free coil of colon may be twisted about its own mesocolic axis, or it may form an axis around which a suitable coil of the lesser bowel may be twisted, just as in the case of the sigmoid flexure.

The symptoms resemble those of volvulus of the sigmoid flexure, but the phenomena, on the whole, are less acute; the distension is less, and the trouble is more or less evidently on the right side.

(*c*) In this variety of volvulus the cæcum has been described as "bent upon" itself or twisted upon itself. In the former instance the cæcum is bent in a plane at right angles to its long axis; the result is that the lower part of the caput coli is found in front of the ascending colon, its posterior surface becomes anterior, while the appendix becomes uppermost. At the angle of the bend there is, of course, a deep crease

across the bowel, and by the bending in of the mucous membrane at this crease the lumen of the gut is occluded.

In the other variety the cæcum is twisted round its own long or vertical axis, so that its relations to the ascending colon are practically undisturbed.

In all these forms of volvulus of the cæcum the gut is imperfectly developed. The colon on the right side is still possessed of an extensive mesocolon, and the cæcum is often undescended or is found about the centre of the abdomen.

The symptoms of volvulus of the cæcum vary greatly, and present almost every phase between acute obstruction of the colon on the one hand, and partial or even chronic obstruction on the other. Diarrhœa has been noted at the outset of the acute cases. Peritonitis is common. The distension of the bowel may be excessive, and the cæcum may become perforated.

C. *Volvulus of the small intestine.*—Twists of this part of the bowel may be considered under two categories. In one a loop of the small intestine is twisted about its own mesenteric axis, in the other a suitable coil or loop of the bowel is engaged in a volvulus with another suitable coil.

(*a*) A volvulus of the small intestine about its mesenteric axis.—The condition of the intestine which favours twisting is identical with that which predisposes to volvulus of the sigmoid flexure. A certain part of the bowel has an unduly long mesentery, whereby it becomes, to some extent, separated from the remainder of the intestine. The two ends of the coil so individualised are brought more or less together, so that a possible pedicle is formed about which the gut may be twisted. This condition of parts may be found in a loop of ileum that has long been herniated and then reduced. The approximation of the two ends of a coil may be brought about by mesenteric peritonitis, due to glandular disease or to other causes. The twist is usually from left to right, and, as a rule, represents one complete turn. Fatal obstruction, however, may follow in instances where the bowel has described but half a turn.

The part of the bowel involved is usually the lower ileum. The symptoms vary considerably. The attack may be indistinguishable from a strangulation by a band, and may end fatally in from thirty-six hours to nine days. On the other hand, the patient may exhibit for weeks the phenomena of a partial obstruction of the lesser bowel, or his illness may be marked by repeated abrupt attacks of acute obstruction from which he repeatedly recovers.

A sudden onset is usual. Diarrhœa is not met with—at any rate was not a feature in the few recorded cases.

(*b*) Two suitable coils of small intestine are twisted together—the one serving as an axis about which the other is wound. The suitability consists in the involved loop being possessed of a long and narrow mesentery, or of the loop that forms the axis being fixed by its extremity to some point on the parietes. This form of volvulus is very rare. It would

appear that the symptoms to which it gives rise are of an acute character, as is often seen in like forms of volvulus where two coils of bowel are involved, one coil being composed of small intestine. It may be remarked that not a few cases recorded as volvulus of the small intestine in no way merit this description.

III. Intussusception.—By the term "intussusception" is understood the prolapse of one part of the intestine into the lumen of an immediately adjoining part.

If an intussusception be viewed in vertical section, it will be seen to consist of six layers of intestine, three on each side of the central canal, all more or less parallel to one another. It will be noticed also that the arrangement of the layers is such that mucous membrane lies in contact with mucous membrane, and peritoneum with peritoneum. On horizontal section the invaginated mass will show three concentric rings of bowel with, of course, the same mutual relations of the mucous and serous surfaces. The outermost of the three layers is known as the intussuscipiens, the sheath or the receiving layer. The innermost cylinder is known as the entering layer, and the middle one as the returning layer. Taken together, these two layers form the intussusceptum. The "neck" of the intussusception is at its upper part, where the returning layer joins the sheath. The ridge formed by the junction of these two layers is known as "le bourrelet." The "apex" of the intussusception is at the lower part of the intussusceptum, where the entering and returning layers join.

Anatomical varieties.—Invaginations may occur at any part of the intestine from the duodenum to the rectum. They may be conveniently divided into three classes: (*a*) the enteric; (*b*) the colic or rectal; and (*c*) the intussusceptions which involve the ileo-cæcal segment of the bowel.

(*a*) Enteric invaginations may occur in any part of the lesser bowel. They are most common in the lower jejunum, and then in the ileum.

It would appear that jejunal intussusceptions bear to iliac intussusceptions the proportion of about four to one. Invaginations involving the small intestine are usually short, and do not involve more than from three to ten inches of the bowel on an average. Most of the "intussusceptions of the dying" are enteric.

(*b*) Colic intussusceptions present many varieties. The ascending colon may be invaginated into the transverse, the transverse into the descending, and the descending colon into the sigmoid flexure. They are most frequently met with in the two last-named parts of the colon. Owing to the comparative fixity of the large intestine it happens that these intussusceptions are usually short; and indeed, taken as a whole, they form invaginations which in point of size are the smallest of the whole series. When the rectum is involved the upper segment of this intestine is invaginated into the lower part. Such intussusceptions must of necessity be short, since in the most extreme cases they must be limited by the length of the rectum itself.

(*c*) The intussusceptions that occur in the ileo-cæcal region may be divided into two main classes, the ileo-cæcal and the ileo-colic.

The ileo-cæcal form is the commonest variety of invagination, while the ileo-colic is the most rare. In the former the ileum and cæcum pass into the colon preceded by the ileo-cæcal valve. The internal cylinder is formed of the termination of the ileum; the external cylinder or sheath is formed by the colon alone, while the apex of the intussusception is represented by the ileo-cæcal valve. This form may attain great size, and not infrequently the valve traverses the whole length of the large intestine, and ultimately presents itself at the anus, or even protrudes through the sphincter.

In the ileo-colic variety the termination of the ileum is prolapsed through the ileo-cæcal valve; the valve and the cæcum remaining for a time at least in their normal situations. The apex of the intussusceptum must always be formed by some portion of the terminal part of the ileum. This intussusception is commonly associated with some secondary invagination of the cæcum and colon itself.

Relative frequency of the various forms.—According to Leichtenstern the different anatomical varieties are thus distributed in 100 cases: ileo-cæcal, 44 per cent; enteric, 30 per cent; colic (including rectal), 18 per cent; and ileo-colic, 8 per cent.

The mode of growth of the intussusception.—In all the forms, with the exception of the ileo-colic, the method of increase is as follows: when an intussusception increases in length after a piece of bowel has been primarily invaginated, the increase is at the expense not of the enteric layer but of the external or receiving layer. For example, let it be supposed that a portion of the termination of the jejunum is invaginated into the ileum; if now the mass increase in length, it will do so solely at the expense of the ileum; no more of the jejunum will actively enter into the intussusception, so that no matter what segment of gut formed the original apex of the intussusception, that apex will remain the same even if the invagination double or treble its original length. In the ileo-cæcal variety the cæcum is turned into the ascending colon, and the valve forms the apex of the intussusception. As the invagination increases, the ascending colon becomes inverted, then the transverse and descending colon, until at length, when the sigmoid flexure is reached, no trace of the ascending nor probably of the transverse colon will be left; but the valve will still form the tip of the intussusception.

In the ileo-colic variety the method whereby the intussusception increases is, in the first instance at least, somewhat different. A portion of the terminal ileum is protruded through the ileo-cæcal valve, and the invagination may increase for some time solely by the prolapse of more and more ileum, the sheath remaining perfectly unchanged. This is exactly the opposite to what happens in other intussusceptions. Sooner or later, however, no more ileum can become prolapsed. The part protruded may become fixed by adhesions; or from congestion or distension of the ileum the valve may offer a rigid resistance to any further invasion

of the colon. In such a case, if the intussusception still continue to increase, it must do so by the method observed in other forms of invagination, namely, at the expense of its sheath. No more ileum can enter, but the cæcum can be turned in, and then the ascending colon, and so on until at last the rectum may be reached.

The intussusception of the dying is a form of invagination that occurs probably a little while before death, and depends upon certain irregular peristaltic movements which may be supposed to occur during the act of dying. It differs very materially from the ordinary or obstructive intussusception.

Intussusceptions of this kind cause no symptoms during life; they are first discovered at the autopsy. They are always very small, are always free from any trace of congestion or inflammation, and interfere little with the lumen of the bowel. With the most trifling amount of traction they can be reduced. They are most usually met with in children, and especially in such as have died of brain disease. They occur in association with perfectly normal viscera. They are uncommon in adults.

The obstructive invagination is usually single; the intussusceptions of the dying are often multiple, and are nearly always confined to the small intestine. The obstructive intussusception is, in direction, almost invariably descending, that is, the in-turning of the bowel wall is in the direction of the anus; the intussusceptions of the dying, on the other hand, are often ascending or retrograde; or the two forms may be present in the same body.

Retrograde, double, and triple intussusceptions.—These unusual forms may conveniently be considered here. I have said already that the common or obstructive invagination is almost invariably descending in direction. To this rule there are very few exceptions. Out of a collection of 593 cases Leichtenstern could find only eight examples of a primary ascending or retrograde intussusception of the obstructive variety.

A primary descending intussusception may be associated with a secondary ascending one, the two occupying the same segment of the bowel. In such cases the retrograde invagination is external to the layers which take a descending direction.

Instances of *double* intussusception are fairly common. In these cases one invagination is primary, the other is secondary. The primary tumour acts as a foreign body in the intestine, and leads to fresh infolding of the walls of the bowel. The secondary invagination concerns only the sheath or receiving layer of the primary tumour. The variety is met with both in the colon and in the small intestine, and in the intussusceptions of the dying as well as in the obstructive forms.

Cases of *triple* intussusception are not so common. Here also there is a primary invagination and then two secondary invaginations, the first of which involves the sheath of the primary intussusception. In these cases it will be evident that the tumour will present no less than seven

layers of intestine in the place of the three met with in the ordinary intussusception.

The general pathological changes in an intussusception.—The part played by the mesentery.—As an invagination increases it is obvious that the mesentery must be drawn in with the bowel. In a tumour of any magnitude it is found between the two layers of the intussusceptum, drawn out into the form of a cone, with its apex at the extremity of the intussusception and its base at the neck.

The traction exercised by the mesentery has a considerable effect upon the tumour. It bends the intussusception so that it becomes curved in outline, the concavity of the curve being towards the mesenteric attachment. Another result of the traction is that the axes of the intussusceptum and of the intussuscipiens do not correspond. The former does not lie in the axis of the latter, but is placed eccentrically nearer to the mesenteric border of the bowel. It follows also that the orifice of the intussusceptum is made to assume the aspect of a slit, and looks not so much towards the lumen of the bowel below as towards the mesenteric side of the receiving layer.

In the colon the mesocolon may play somewhat the same part as the mesentery. In colic intussusceptions, however, it is very common to find the several layers of the mass parallel to one another, the aperture being in the centre and directed towards the central axis of the gut below.

Intussusceptions of the rectum are all more or less free from curving.

How obstruction, strangulation, and irreducibility are produced.—Mere invagination of the bowel need not lead of necessity either to strangulation of the involved part, or to complete or even serious obstruction to the lumen of the intestine.

The part, however, in acute cases may become strangulated, and, as a result, the whole of the intussusceptum may become gangrenous.

Obstruction to the passage of matters along the intestine may be brought about in many ways.

(*a*) The orifice of the intussusceptum is rendered slit-like by the dragging of the mesentery, and may be opposed to the wall of the receiving layer.

(*b*) The intussusceptum may be so bent or curved upon itself as greatly to narrow the lumen of the inner cylinder.

(*c*) The considerable thickening that the tunics of the involved bowel undergo, as the result of congestion, exudation, and inflammation, tend greatly to narrow the lumen of the passage.

(*d*) The already narrowed passage may be finally occluded by a foreign body, for example undigested rice, plum stone, etc., or by blood clot, or by a polyp attached to the bowel.

The question of the irreducibility of the invagination is one of extreme importance in the prognosis. If the intussusception be irreducible, then cure by spontaneous reduction is impossible, as also is reduction by means of forcible enemas or by laparotomy. On the other hand, if the tunics of the mass be glued together by adhesions about

the neck, the parts are most favourably placed for spontaneous recovery by elimination of the gangrenous intussusceptum.

The irreducibility very commonly depends upon adhesions which vary in situation. Sometimes they are limited to the neck of the mass, at other times to its apex, while in a third class of cases they involve the whole length of the inner and middle layers. On the whole, the last named are the most common, although adhesions limited to the neck of the intussusception are probably the more usual in acute cases.

In extensive invaginations it is common to find the first few inches of the intussusception fixed by adhesions while the remainder is quite free. In these cases it is probable that the adherent parts represent those first invaginated, no adhesions having formed between the layers subsequently prolapsed. Thus it happens that the whole intussusception, with the exception of the last inch or so, can be readily reduced.

In examples of chronic intussusception adhesions are the rule; they are present in about 80 per cent of the cases. In acute invaginations adhesions are as often absent as present. The earliest time for the appearance of definite adhesions is the third day. It is needless to observe that recent adhesions are very soft and yielding, so that in acute examples, although false ligaments may exist, they need not in themselves offer any serious obstacles to attempts at reduction.

Irreducibility, however, may depend on other causes than the results of local peritonitis.

(α) The swelling of the intussusceptum may be so excessive as entirely to prevent reduction.

(β) Since the swelling and thickening of the coats are most apt to affect the convexity of the intussusceptum, it often happens that so curved an outline is given to that part, and so great an alteration effected in its density that for this reason also reduction is quite impossible.

(γ) The invaginated bowel may become peculiarly twisted, and may thus be rendered irreducible.

(δ) In ileo-colic invaginations an especial obstacle to reduction is offered by the ileo-cæcal valve.

(ϵ) When a polyp exists at the apex of the intussusceptum, it forms, when associated with swelling of the gut above it, a very definite impediment to reduction.

Changes in the bowel.—The bowel above the intussusception shows in acute cases no gross changes other than those of dilatation and congestion; in chronic forms, however, its walls are usually hypertrophied, and in some instances this hypertrophy has attained considerable dimensions.

Ulceration of the intestine above the involved segment is comparatively rare, and is somewhat more common in chronic than in acute cases. Perforation may occur as a result of this ulceration.

The sheath or receiving layer seldom shows any gross changes. It may be congested or a little thickened, it may be much wrinkled and thrown into many folds, and it may be the seat of some local peritonitis.

This layer not infrequently presents ulcerations of its mucous membrane, which are often multiple and may lead to perforation, or a part of the wall of the sheath may become gangrenous.

With regard to the changes in the intussusceptum the cylinders involved become distended with blood, and hæmorrhages may occur in their substance or from their surfaces. It is from the latter source that is derived the bleeding which is so often a conspicuous feature in intussusceptions, especially those of an acute course. The walls may become rapidly œdematous and greatly swollen, and this condition runs on readily to gangrene. In more chronic cases great thickening of the layers of the intussusceptum may be met with as a result of long-continued congestion and insidious inflammation of a low type. In both acute and chronic cases the thickening of the layers may be equally distributed throughout the involved cylinders, but far more usually it is most conspicuously marked in two places, namely, at the apex of the intussusceptum and along its convexity.

One of the most important and constant changes in the intussusceptum is gangrene. This condition is met with both in acute and chronic cases, although it is always more common, and usually more extensive, in the former. It may involve the whole mass of the intussusceptum, which may separate at the neck and be discharged from the bowel. This occurs, as a rule, in acute invaginations, although it is sometimes met with in chronic cases that end acutely. The gangrenous part which is eliminated may vary in length from a few inches to several feet. The gangrene usually appears first and remains most advanced in the middle layer. Thus it happens that, when the separation of the intussusceptum occurs, the middle cylinder may be disintegrated and in some parts missing; while the entering layer, although dead, may be sufficiently well preserved to show the structure of the bowel. Sometimes the anatomical details of the part are singularly well preserved in the separated intestine. An example of this is afforded by a specimen in Guy's Hospital Museum, showing the cæcum and the whole of the ascending colon, which were passed on the eleventh day; the patient recovered. Sometimes, however, the inner cylinder is more extensively involved in the gangrenous process than the middle layer. This condition is usually met with in the ileo-cæcal invaginations, where the part of the intussusceptum formed by the small intestine may perish before that segment formed by the large.

Speaking generally, then, it may be said that in acute invaginations gangrene is more common and extensive, that it involves principally the neck of the mass, and is associated with an elimination of the cylinders more or less in their entirety. In the chronic forms the gangrene is less rapid, is most marked at the apex, and leads usually to a slowly progressing destruction whereby the intussusceptum is eliminated in fragments.

Causes of intussusception.—With regard to the exciting causes of intussusception—and with this feature in the etiology we need only be concerned in this section—the following facts may be mentioned:—

Probably in about 50 per cent of the cases no cause for the invagination can be found. Many of the subjects of the affection, however, are described as delicate and anæmic, or as wasted. In some 10 per cent there is a history of bowel troubles—of enteritis or diarrhœa or of marked intestinal irregularity. In other 10 or 15 per cent there is the association of a mass of undigested food which may have produced definite colic. Intestinal polypi are credited with producing 5 per cent of the invaginations—which is probably too high an estimate. Injuries and exposure to cold and certain acute and chronic maladies, such as the eruptive fevers, whooping-cough, and hernia, are credited with some efficiency in the production of the trouble.

IV. Stricture.—The name stricture is conveniently limited to a narrowing of the lumen of the bowel brought about by changes in the coats of the bowel itself. These changes depend practically upon two conditions only, upon cicatrisation after loss of substance and upon carcinomatous deposits.

A third form of stricture is congenital and depends upon some defect in development; it most commonly occurs in the rectal and anal region, The congenital strictures which concern us at present are most often met with in the small intestine, and are most common about the lower end of the ileum. The stricture may be complete or incomplete, single or multiple, or a considerable part of the bowel may be actually wanting. After the lower end of the ileum the next most common place for a congenital stricture would appear to be the beginning of the jejunum.

Congenital stricture (A.) plays but a very small part in practical medicine; and the forms which call for our consideration in detail are: B. the cicatricial, and C. the cancerous.

B. *The cicatricial stricture.*—This depends upon the contracting of a scar consequent upon loss of substance by ulceration or limited gangrene of the inner coats. The aspect and degree of the stricture will obviously depend upon the situation and extent of the original loss of substance. A limited patch of ulceration placed in the long axis of the bowel may lead to very insignificant narrowing of its lumen, while an ulcer no more extensive, but disposed transversely around the gut, may produce an annular constriction that may almost close the tube. Some contracting scars may merely alter the course or direction of the bowel, while an unequally contracting scar may produce obstruction as well by actually diminishing the size of the canal as by distorting the intestinal walls.

It is convenient to divide the cicatricial strictures into three classes:—(*a*) Those depending upon primary ulceration; (*b*) those that are subsequent to lesions following strangulated hernia; (*c*) those that may follow injury. The first class concerns both the large and small intestine; the others, so far as the cases I have collected serve to show, concern only the lesser bowel.

(*a*) The forms depending upon ulceration are the following:—

(α) Typhoid ulcers.—These ulcers lead to distinct and recognisable scars, but it is only in extremely rare cases, and probably when the ulcers have been chronic, that they produce any stenosis of the intestine. Many of the cases ascribed to enteric fever will not bear examination.

(β) Dysenteric ulcers are frequent causes of stricture. The cicatrices often extensive, rigid, and dense. The contraction may be very irregular. The gut may be much puckered, or thrown into irregular folds or in other ways distorted.

I believe that not a few instances of so-called "scirrhus" of the colon are really examples of dense, hard, dysenteric scars, associated with much contraction and with firm warty excrescences. Dysenteric strictures are most often met with in the rectum, sigmoid flexure, and descending colon. They are fairly common, also, both at the hepatic and the splenic flexures.

(γ) The catarrhal ulcer—the product of acute or chronic catarrh—is credited with the occasional production of stricture, the lesion being usually in the colon.

(δ) Syphilitic ulcers of the bowel are most often due to the breaking down of gummas in the submucous tissue. Excluding the rectum this form of ulcer is said to be most common in the lower ileum.

(ε) Tuberculous ulcers may assume an annular form and lead to stenosis. These ulcers may be met with in almost any part of the intestine, and often involve a great extent of the bowel; they are most common however, in the lower ileum and about the ileo-cæcal valve. When associated with marked tuberculous deposits and with tuberculosis elsewhere they seldom heal. The milder cases, however, cicatrise, and then the annular ulcers may produce some stenosis of the intestine; this stenosis is usually, I think, of a moderate degree.

As regards locality, cicatricial strictures of the lesser bowel, taken generally, are usually situated in the ileum, and preferably in the middle and lower parts of the ileum. In the colon about 50 per cent of these cicatricial strictures are in the sigmoid flexure. Next in frequency come the descending colon and splenic flexure, and beyond these parts the stenoses become rarer and rarer as the cæcum is approached.

In comparing the large intestine with the small, one is struck with the fact that the simple stricture of the colon is nearly always single. On the other hand, out of ten recorded cases of cicatricial stricture of the lesser bowel there were six instances of single stricture and four of multiple.

Certain of the multiple strictures found in young adults are no doubt of congenital origin.

(b) Stricture after strangulated hernia.—The stricture which may form in a piece of the intestine that has been involved in a strangulated hernia is due to cicatrisation, and follows upon ulceration or limited gangrene of the involved bowel. I have found six recorded examples of this stricture, besides specimens to be seen in some of the London museums. It has followed upon both inguinal and femoral rupture, and

has produced symptoms of obstruction at a period (after the relief of the hernia by operation), varying from one month to "some years." In three cases the ileum was involved, in one the jejunum. In one instance $1\frac{1}{2}$ inch of the bowel was found contracted and thickened. In other examples the stricture was of a very limited extent and annular, as if a narrow tape had encircled the bowel.

(*c*) Stricture after injury.—I find records of two cases of stricture that were evidently due to cicatrisation following injury to the bowel. Both patients were men aged about forty-five. In one case symptoms of obstruction came on three months after the patient had been ridden over; in the other case four months after a blow upon the abdomen. In the former the ileum was involved, in the latter the upper jejunum. Both strictures were very narrow, and adhesions existed in the vicinity of the stenosed segments.

B. *The cancerous stricture.*—Carcinoma of the intestine may be either primary or secondary. As a secondary growth it may appear either by metastasis or by extension from neighbouring parts. So far as practice is concerned, the growth causing obstruction or definite intestinal symptoms is usually primary; the metastatic form need not be considered here.

There would appear to be but one form of primary cancer met with in the intestine, and that is the cylindrical-celled epithelioma. Such a growth may undergo colloid degeneration and so produce a "colloid cancer."

Epithelioma may be met with in the bowel under three different aspects: (*a*) As small nodules; (*b*) as flattened plaques involving only a portion of the circumference of the gut; and (*c*) as annular deposits which surround the bowel like a ring. Into the microscopic characters of this form of epithelioma it is not necessary to enter. The morbid changes begin in the epithelium of Lieberkühn's glands, and produce at first a great thickening of the glandular layer of the mucous membrane for some distance; or the new growth may be limited to one spot and a projecting nodule be produced. Some of these nodules form conspicuous tumours which are apt to assume a polypoid outline. In other instances the growth spreads laterally rather than towards the lumen of the bowel, and an epitheliomatous plaque is produced. In this form a raised flattened mass of cancer is formed upon one part of the intestinal wall. Its edges are well defined and often abruptly raised, its centre uneven and ulcerated.

The commonest form, however, under which epithelioma of the intestine presents itself is that of an annular band around the intestine. Compared with this aspect of the growth the nodules and plaques may be said to be comparatively rare. It is this form of epithelioma that is usually met with under the title of malignant stricture of the intestine, and it probably represents the only true cancerous stricture of this part. The appearance of these strictures is characteristic. The gut at the stenosed part appears to be suddenly constricted, as if a piece of

cord had been drawn very tightly about it. The stricture is usually quite annular but insignificant in width, comparatively little of the gut, as measured along its long axis, being involved. The peritoneum about the stenosed part is often thickened, and the bowel itself is not infrequently adherent. On examining the gut from the inside the stricture may appear as an annular and contracted deposit, the surface of which is irregularly ulcerated.

Strictures of this character are often very narrow. They are much more common in the large than in the small intestine, and I have met with several specimens where the margins of the ileo-cæcal valve were the seat of an epitheliomatous growth.

It is important to recognise the fact that epitheliomas of the intestine appear as single growths. The gut above the stricture becomes dilated and its walls hypertrophied. In long-standing cases this hypertrophy may be considerable and far spread. Thus in stricture of the sigmoid flexure it may reach to the ileum.

The distension of the colon above a stricture may be very great. In a case of epithelial cancer of the sigmoid flexure causing stricture, reported by Dr. Fagge, the splenic flexure of the colon was found to be as large as a distended stomach.

The mucous membrane of the bowel above the obstruction is usually ulcerated, and perforation caused by these ulcers is a common cause of death after stricture. In the small intestine the ulceration is, as a rule, situated just above the stenosed part, and if perforation occur it will occur here.

As regards the colon, the whole of its mucous membrane above the stricture may be ulcerated, but as a rule the ulceration is much more narrowly limited. When the stricture is at some distance from the valve, ulceration may be noted in two distinct places, namely, just above the obstruction and in the cæcum, the intervening mucous membrane being quite healthy.

The perforating ulcer above the stricture need not open into the peritoneal cavity. In a few cases where adhesions have formed the perforation has been so placed as to give temporary relief to the obstruction. Thus in one case of stricture of the valve, the ileum opened into the first part of the colon, forming a fistula bimucosa through which fæces could pass. Other cases of relief by the formation of such a fistula have been reported; also instances where the colon above a stricture in a distorted sigmoid flexure was found to have opened into the bladder or rectum.

The ulceration above a malignant stricture of the more fixed parts of the colon may lead to an abscess around the gut. This abscess may discharge externally and lead to a fæcal fistula; or after such discharge it may heal, though the healing is often but temporary.

The condition of any stricture in its relation to the clinical aspect of the case.—The stricture at the time of death may be wide enough to admit the tip of the forefinger; on the other hand, it may be so narrow

that water will merely trickle through it in drops, or it will admit only a probe or a goose-quill. As a rule the narrowest strictures are met with in the small intestine. A stricture can contract to narrow dimensions without producing a rapidly fatal result if the contents of the part of the bowel that it involves be fluid. This is one reason why strictures may be narrower in the small than in the large intestine.

The precise manner in which a stricture of the intestine brings about the death of a patient is by no means the same in every case. In some instances the stricture becomes narrower and narrower, the obstruction becoming by slow degrees more and more complete, until at last, after following a chronic and lingering course, it causes death. In other cases the stricture, having obstructed the bowel to a certain extent, appears to undergo no further contraction, but the patient dies worn out by the long-continued abdominal troubles, or succumbs to an increasing marasmus. There are cases which linger for a while, and then end somewhat more abruptly; that is to say, for some considerable time the malady may present the symptoms of a chronic obstruction, and the fatal issue be brought about by an attack of acute obstruction. Instances of this kind depend upon many different pathological conditions. Thus a plug of hard fæcal matter may have blocked up a stricture that had of itself caused no very serious amount of obstruction; or this blocking of the stenosed part may have been brought about by some foreign substance. In other instances folds of mucous membrane from the gut above the stenosed part may so fall across the orifice of the stricture as to close it like a valve. In these cases water may be injected with ease from below, but only with much difficulty from above. To cases such as these must be added that extensive series where the small intestine at the seat of the stricture has become so bent as to have its lumen more or less abruptly occluded, or where "kinking" has occurred, or where the narrowed bowel has become still more occluded by adhesions and by matting of its coils together.

In stricture of the sigmoid flexure, moreover, an acute termination to the case by the production of volvulus or of acute bending is by no means uncommon.

Position and relative frequency of the different forms of stricture.—Stricture of the colon is comparatively common; stricture in the small intestine is undoubtedly rare.

One hundred examples of stricture of the small intestine may probably be divided as follows:—

Due to cicatrix after ulcer	44
,, ,, injury	8
Following strangulated hernia	16
Due to carcinoma	30
Of congenital origin	2

Cases of stricture of the ileo-cæcal valve are more often due to malignant growth than to cicatrix.

One hundred examples of stricture of the colon may be divided as follows:—

Due to malignant growth	68
Due to simple stricture	26
Cause unknown	6

The situation of the stricture in the colon in 100 cases is as follows:—

Sigmoid flexure	58
Descending colon	11
Splenic flexure	7
Transverse colon	7
Hepatic flexure	9
Ascending colon	2
Cæcum	6
Total	100

V. Obstruction by tumours and foreign substances within the bowel. — In addition to the carcinoma already described the following growths may obstruct the bowel. In all instances they grow from the wall of the bowel itself.

(*a*) *Adenoma or polyp.*—These grow from the mucous membrane, and appear to have their origin in the follicles of Lieberkühn. They present on section a number of tubes, passages, and spaces, all lined with columnar epithelium, and supported by connective tissue which may vary in structure from a lax myxomatous meshwork to a substantial fibrous substance. It is upon the character of this supporting tissue that the physical characters of the growth in some part depend, the laxer tissues forming soft, and the denser structure firm, polypoid masses.

These adenomas are most frequently met with in the rectum and colon, and form the commonest variety of benign growth. They frequently occur in children, and are perhaps more often multiple than single.

(*b*) *Fibroma.*—These arise from the submucous tissue. They are nearly always numerous and small, and usually appear as sessile nodules. They may be pedunculated. A large number of growths are described as "fibrous polypi," but trustworthy microscopic evidence is wanting to show that even the majority of these are composed of fibrous tissue.

(*c*) *Fibromyoma.*—Several examples of this kind of benign growth have been placed on record, the nature of the tumour having been verified by microscopical examination. They arise from the submucous and muscular coats, and have an arrangement of parts like that seen in simple fibromyoma of the uterus. Those having origin from the submucous coat spring, no doubt, from the mucosa muscularis. They are small.

(*d*) *Lipoma.*—These growths spring from the submucous layer, take a polypoid form, are often multiple, but seldom of great size.

(*e*) Among the still rarer growths may be mentioned *angiomas*, examples of which have been described by authors. Some of these are probably very vascular fibromyomas.

Rokitansky has described cases where multilocular cysts filled with serum were found partly embedded in the intestinal wall. It may be that these were cystic adenomas.

Considered collectively, benign tumours of the intestine are usually met with in the form of polypi. As such they may have very distinct pedicles. In shape they are round, oval, or pear-shaped. In size they vary from the dimensions of a pea to that of a small orange or a pear. They are usually covered by normal mucous membrane. As regards their place of origin, the great majority, probably not less than 80 per cent, are met with in the rectum; next in frequency come those in the ileum and then in the colon. They are rare in the jejunum, and still rarer in the duodenum. In the small intestine the favourite site is the lower extremity of the ileum.

The growth is usually attached to the convex border of the gut, or at least away from the mesenteric border.

Benign polyps are often very numerous. The occurrence of three, four, or five polyps in the same division of the bowel is quite common.

Benign growths of the intestine may give rise to no symptoms during life, and may even attain large size and become numerous without affording any evidence of their existence.

Polyps most usually cause symptoms when in the rectum, producing tenesmus, bleeding from the bowel, difficult defæcation, and a sense of a foreign substance in the gut. The same symptoms in a less marked degree may attend growths arising from the sigmoid flexure.

In other parts of the intestine the polyp usually causes obstruction, if it occlude the gut at all, by inducing an invagination; this is particularly the case with such as grow from the ileo-cæcal valve and from the terminal part of the ileum. Benign tumours have also produced intussusceptions in other parts of the bowel, in the rectum, in the sigmoid flexure, and in all parts of the colon.

When the mass is of large size, or when the growths are multiple, symptoms of obstruction may be produced which more or less closely resemble the symptoms of stricture, save that they are usually more chronic and for a while at least less marked.

So far as I am aware, it would be impossible to diagnose cases of obstruction due to simple neoplasms from cases of stricture.

In a few cases the polyp has separated from its attachment and has been passed per anum. This mostly occurs in connection with such growths as spring from the rectum or sigmoid flexure; although I am disposed to believe that some reported cases, where strange fleshy masses have been passed with motions, have been examples of the spontaneous removal of a polyp.

(*f*) *Sarcoma.*—Sarcomatous tumours have been met with in the intestine both as primary and as secondary growths. They are usually of the spindle-celled variety, and are seldom of the small-celled kind. They very rarely, indeed, appear as polyps, but rather tend to spread

around the bowel, and then probably produce one form of what is vaguely known as a "cancerous" stricture.

Several examples of *lympho-sarcoma* of the intestine have been recorded in connection with Hodgkin's disease (see vol. iv.). The neoplasm in these cases appears in the adenoid tissue of the gut and in Peyer's patches, and may attain considerable dimensions. At many parts in such cases the whole calibre of the gut has been surrounded by an extensive morbid growth, while in other places only portions of the intestinal wall have been invaded. Their most remarkable feature is, that, so far as recorded examples at present show, obstruction is not usually produced. Indeed the subjects of the disease appear to have had either no special abdominal symptoms or else a more or less pronounced diarrhœa. Dr. Carrington has reported a case where a lympho-sarcomatous mass weighing no less than half a pound occupied the cæcum, and yet no symptoms of obstruction were produced; nor indeed does special attention appear to have been directed to the abdomen during life.

Obstruction by foreign bodies.—By a "foreign body," as applied to the intestinal tract, is meant any substance that can resist the digestive action of the fluids of the stomach and bowels.

These foreign bodies may be conveniently divided into three classes: 1. Rounded, or irregularly shaped substances which may be capable of passing readily through the intestine; among such are pebbles, stones, fruit stones, coins, bullets, and the like. 2. Sharp-pointed bodies and substances of irregular shape which may readily catch in the mucous membrane, or are of an outline that would favour their becoming fixed in the alimentary passages; such are pins, needles, hooks, plates carrying false teeth, pieces of bone, pieces of metal or of porcelain, nails, screws, and other such substances. 3. Indigestible materials of small size which are apt to accumulate until they form huge masses; they are composed of husks of the oat, vegetable fibres, and grape skins; or of hairs, wool, or yarn.

There is no doubt but that the majority of the foreign substances that are swallowed are in time passed by the anus. Most of those placed in the first of the above classes would be so evacuated in the course of a few days, or even after forty-eight hours. Others would be retained for a week or a fortnight or longer without causing inconvenience. Many foreign substances which may be placed in the second class have also been passed with comparatively little inconvenience. Some such bodies have lingered in the alimentary tube for weeks, for months, and even for years. Mr. Pollock quotes a case where a plate carrying six false teeth was swallowed and passed at the end of three days. In another like instance where the plate held four teeth the mass was evacuated per anum at the end of six months.

The most remarkable example of foreign bodies in the alimentary canal is afforded by the case of the "human ostrich," reported in the *British Medical Journal* for May 5, 1894, where the collection of foreign substances numbered over 100.

When the foreign substance is not passed by the natural channels,

it is apt to remain lodged in certain special parts of the tube, namely, in the stomach, the duodenum, the lower end of the ileum, the cæcum, or the rectum. Of all these situations the cæcum is the one in which lodgment is most likely to take place.

As a foreign body passes along the canal it may cause obstruction at any point, and that obstruction may prove fatal. The progress of the larger and more irregular substances is marked by pain, and by attacks of temporary obstruction, associated with colic, vomiting, and constipation. In other instances an impacted foreign body has given rise to long-continued symptoms of partial obstruction, symptoms which may become very chronic yet never very severe.

There is plenty of evidence to show that these bodies may remain for weeks, months or years in the stomach or in some part of the intestine without causing active mischief, but that, when so lodged, they may almost at any time induce changes leading to a fatal result. Even when they have been long retained they may be discharged safely by the natural passages. Thus in one of Mr. Pollock's cases, a plate carrying false teeth had been swallowed, and after remaining in the stomach for ninety-seven days was finally ejected by vomiting. The impacted foreign substance, however, is very apt to induce some ulceration of the mucous membrane; this may lead to perforation and to fatal peritonitis, or some local chronic peritonitis may be excited in the part lodging the substance, and the gut may become narrowed thereby. Such narrowing may increase after the evacuation of the body, and may lead ultimately to obstruction.

In another class of cases the ulceration of the mucous membrane leads to the formation of a fistula through which the foreign body may be discharged. This fistula may communicate with the exterior; or the fistula may form between the stomach and the transverse colon, or between the ileum and the colon, or even between the coil lodging the foreign body and the rectum or the vagina. By such fistulous channels foreign bodies have been evacuated after long intervals.

Small sharp-pointed bodies, such as needles, may readily penetrate the intestine and work their way to the surface, where they may be recognised and removed. Thus I once extracted from under the skin of the groin a needle that had been swallowed by a child some months previously. Metallic substances in the alimentary canal may be rendered visible by means of the Röntgen method of photography especially in children.

The foreign bodies of the third class that cause obstruction by accumulation may form immense masses. When in the intestine they may lead to chronic and fatal obstruction, or may induce chronic or acute peritonitis. Thus Marshall mentions an occlusion of the duodenum by a pound of pins which had been swallowed. In an instance quoted by Duchaussoy in his memoir the obstructing mass was composed of 700 cherry-stones. In a case recorded by Dr. Quain the mass consisted of four pounds of cocoa-nut fibre.

Obstruction by gall-stones.—The lumen of the intestine may be obstructed at certain points by a gall-stone which has entered it from the gall-bladder, and is passing along its way to be discharged at the anus. In the great majority of cases the gall-stone passes along the intestine without any difficulty, and, indeed, without exciting symptoms of any kind. The instances where obstruction, whether temporary or permanent, is thus produced must be regarded as exceptional. It may be surmised that a stone that will pass along the narrow and somewhat rigid bile-duct cannot be expected to meet with any obstruction in the intestine. Even the lumen of the ileo-cæcal valve is many times greater than is that of the common duct. But the gall-stones that cause occlusion do not enter the intestine by the biliary passage; they enter by means of a temporary fistulous communication between the gall-bladder and the duodenum. In very rare examples the communication has been between the gall-bladder and the colon at the hepatic flexure.

As to the size of the calculus which may cause occlusion it must be noted that stones of considerable dimensions have been spontaneously evacuated. Thus stones have passed the anus measuring 2½ inches by 1½ inch, and presenting a circumference of 3½ inches. The stones that have been found impacted in the bowels have in many instances attained considerable dimensions. As examples I might mention the following: a stone measuring 4½ inches by 2¼ inches lodged in the upper part of the jejunum; one with a circumference of 3⅜ inches impacted itself in the lower jejunum; one 2 inches in length and with a circumference of 4 inches, also in the jejunum; and another one inch in length and with a like circumference was impacted in the ileum.

The point in the intestinal tube at which the stone lodges is most frequently in the lowest part of the ileum, or in the duodenum and commencement of the jejunum. An examination of thirty-two cases by Leichtenstern gives the following result:—

In the duodenum and jejunum	10 cases
In the middle of the ileum	5 ,,
In the lower part of the ileum	17 ,,
	32

Obstruction by intestinal stones.—Intestinal calculi or enteroliths may be divided into three classes:—

(*a*) Concretions formed in great part of phosphate of lime, or of phosphate of magnesia, or of the triple phosphate; or stones formed of mixtures of these salts. The concretion is usually single and of small size. It is seldom larger than a chestnut, although a few isolated instances of larger stones have been recorded. In Leichtenstern's list of such calculi three are included, the circumferences of which are 4½, 7½, and 9 inches respectively.

(*b*) Enteroliths of low specific gravity and of irregular form that are porous in appearance and have the consistence of compressed sponge. They are composed mainly of densely felted masses of vegetable fragments

mixed with particles of fæcal matter and with a certain amount of calcareous material similar to that met with in the previous species of stone. These concretions comprise the "oat-stones" or avenoliths which are composed of the indigestible fragments of oatmeal.

In a case by Dr. Down, fatal obstruction was caused by a stone-like mass the size of a hen's egg which had become impacted in the lower ileum. It was composed of densely-packed cocoa-nut fibres, and had probably been formed in the stomach and then passed into the bowel. The patient had been engaged in mat-making.

(*c*) Concretions formed of insoluble mineral matters that have been swallowed as medicines. These are most frequently composed of magnesia. In a case recorded by Mr. Hutchinson a huge mass with a circumference of at least 15 inches was felt in the rectum. Its surface was hard and rough like an oyster shell. It was broken up and removed at several sittings, and it was found to be composed of magnesia and iron, with some earthy matters and many thousands of strawberry seeds. The patient had been in the habit of taking large doses of carbonate of magnesia and of iron.

Enteroliths are most commonly found in the colon and with especial frequency in the cæcum. In the colon they often occupy the sacculi of the gut. They are often met with also in the rectal ampulla, and more rarely in the ileum and in true and false diverticula.

VI. Obstruction by the pressure of tumours and other diseases external to the bowels.—Tumours of various kinds and even displaced viscera may press upon some part of the intestine and cause thereby an occlusion of its lumen.

In the majority of the cases this compression has been effected by a tumour originating in the pelvis.

Thus the bowel may be compressed by a retroverted or retroflexed uterus, or by malignant or other tumours growing from the uterus, or by ovarian tumours of any kind. Among other causes of pressure upon the gut may be mentioned subperitoneal tumours, tumours of the mesentery or omentum, various tumours of the kidney, psoas abscesses and abscesses about the cæcum, hydatid cysts, enlarged spleens.

With regard to the segment of the intestine involved in these cases, the rectum, as it may be supposed, is the part which most frequently suffers. The parts which are involved next in frequency after the rectum are the sigmoid flexure and the lower part of the ileum, then the duodenum and ascending or descending colon. The more mobile parts of the bowel, such as the jejunum and the transverse colon, are practically exempt from this form of obstruction.

VII. Fæcal accumulation.—This subject is dealt with in another article, *vide* p. 700 of this vol. The conditions which favour fæcal accumulation are very numerous, and among them may be mentioned—chronic dyspepsia and constipation, the consumption of masses of indigestible food, such as nuts, irregular meals or hurried eating, loss of muscular power in the bowel due to age, to weakening diseases, to neglect of the bowels, or

to that exhaustion of the gut which may attend the habitual use of aperients. Many of the patients are edentulous and cannot masticate their food. Many are nervous subjects who suffer from that form of intestinal indigestion which is common in the neurotic.

Not a few cases depend upon nervous lesions, the impression conveyed to the bowel being either direct or reflected. Such cases are illustrated by the instances of fæcal accumulation after injury of the spine or associated with grave disease of the cord, and by the local accumulation which may be seen in a segment of the colon, of which the nerves are pressed upon by some tumour, such as an enlarged kidney. The reflex cases are illustrated by the constipation which may depend upon a diseased appendix or an inflamed ovary, and which will vanish when the affected organ is removed. In some instances the bowels share in a general condition of atony.

The condition may be encouraged by damage to the bowel from ulceration—such as that of dysentery, by old adhesions, by unusual bends and folds in the colon—especially in the transverse colon—the outcome of long-continued over-distension. The transverse colon in such circumstances may be bent into a V-shaped figure, the apex of the V or centre of the transverse bowel reaching to the pelvis.

Another cause is a very elongated sigmoid flexure which hangs in the pelvis, and the extremities of which are nearly approximated, as is described in the section on Volvulus (page 815).

Fæcal masses lodged in the colon fill up the sacculi, and tend to become nodular in consequence. They harden by absorption of their more fluid parts, and may attain very considerable dimensions. They are met with most usually in the cæcum or sigmoid flexure, or in the transverse colon.

The bowel containing them is apt to become ulcerated, partly from pressure and partly from the chemical irritation of the highly decomposed mass. These ulcers are most common in the cæcum, because it is upon that part of the colon that the greatest strain comes in fæcal accumulation. They are called "stercoral ulcers," and may be both numerous and extensive. They are usually superficial, but occasionally lead to perforation. If the colon "gives way" as the result of obstruction, the rent or perforation is usually in the cæcum, which, as just stated, bears in all these cases the major part of the strain.

The bowel above a fæcal mass is greatly distended, while below it is more or less contracted.

Fæcal tumours may exist unchanged for weeks or months, and often are the cause of errors in diagnosis.

SYMPTOMS OF INTESTINAL OBSTRUCTION.—From a clinical point of view cases of intestinal obstruction may be conveniently divided into three classes:—I. Acute obstruction; II. Chronic obstruction; and, III. Cases in which symptoms of acute obstruction supervene on those indicative of chronic obstruction. In the paragraphs which follow these three

clinical varieties of obstruction are dealt with in brief outline. The more detailed account of each form will be found on pp. 846 and 860 respectively.

I. Acute intestinal obstruction.—The attack is sudden in *onset*. In the majority of instances no exciting cause is apparent. The patient is seized with very severe abdominal *pain*. This is generally localised about the umbilicus. Sometimes the pain corresponds to the seat of the obstruction, but not commonly. The patient may be "doubled up" by it or roll in anguish on the floor. The pain is usually constant, although liable to exacerbations. There is at first, at least, little or no *tenderness* of the abdomen. There is *collapse*, with great depression of strength, pallor, sunken eyes, a feeble rapid pulse, a cold sweat over the face, a sighing respiration. *Vomiting* appears early, is first composed of the contents of the stomach, is then bilious, and later is brownish and offensive; it is copious and persistent, gives little or no relief, and generally in time becomes stercoraceous. *Constipation* is usually absolute from the first. The belly becomes more or less *distended*, and towards the end of the case is apt to become tender. The *tongue* is foully coated. *Thirst* is intense. The *temperature* is below normal. The *amount of urine* is diminished. If unrelieved, the symptoms persist, the exhaustion increases rapidly, the tongue becomes dry and brown, the face has an aspect of horrible anxiety, the features are pinched, and the eyes sunken. The patient dies with those symptoms of *septic poisoning* which mark the termination of acute peritonitis. There may be delirium, but as a rule the patient retains consciousness to the last. The vomiting usually remains the most distressing symptom.

The majority of the acute cases end in death if unrelieved within six or seven days. The varieties of acute obstruction are enumerated on p. 796.

II. Chronic intestinal obstruction. — The *onset* in this form of obstruction is gradual, and the progress of the malady irregular. There are attacks of abdominal *pain* which are not severe, which come on at first at long intervals, are often provoked by food, and are frequently ascribed to indigestion or colic. These attacks become more frequent, more severe, and of longer duration. They are attended with some vomiting and constipation, and with more or less constant uneasiness within the abdomen. The *vomiting* is probably slight, and does not persist; it is neither copious nor frequent, but much nausea and disinclination for food may remain. The *constipation* at first is not absolute; in the early stages the patient is relieved by aperients. These drugs then act with less and less effect, and at last only occasion severe pain and vomiting. Sometimes there is a period marked by *diarrhœa*. This is a "spurious" diarrhœa due to catarrh excited in the bowel by retained fæcal matter above the obstruction; it is especially met with when the stenosis is somewhat low down in the colon. Between these attacks the patient may feel fairly well, and suffer only from some abdominal distension, much irregularity of the bowels, nausea, malaise and loss of appetite.

The *tongue* becomes white and coated, and the breath often most offensive. The *temperature* is not usually disturbed, nor is the *amount of urine* abnormal. The belly becomes more and more *distended*. A *tumour* is often discovered. Evidence of an *accumulation of fæces* is often present. *Visible coils of intestine* can be seen in movement through the apparently thinned abdominal parietes. When the movement takes place the patient has pain. There are frequent rumbling and *gurgling sounds* in the abdomen, which are very audible to those around. The pain becomes more continuous and more severe, the vomiting is more persistent, the constipation is at last almost absolute, the distension of the belly increases, and the strength rapidly fails.

Unless some accident, such as perforation, occur, the patient (if unrelieved) dies exhausted and marasmic, worn out by the continued pain and vomiting, wasted by inability to take food, and poisoned by the absorption of noxious matter from the horribly putrid contents of his own intestine. The breath has often a perfectly fæcal odour at last.

The phenomenon termed *ballooning of the rectum* has been said to be a sign of stricture of the colon. On introducing the finger into the anus in a case in which this condition exists, the bowel will be found empty, and the rectum so dilated that at first the finger can scarcely reach the limits of its walls; and the walls when felt are perfectly smooth. The condition is not due to mere distension with gas, and the term "ballooning" is therefore unfortunate. The precise cause of this curious condition has not yet been defined. It evidently concerns the innervation of the bowel, and it disappears under an anæsthetic. Ballooning of the rectum is often met with indeed in stricture of the colon, but it is not diagnostic of it: it is not present in every case on the one hand, and on the other it may be met with in localised peritonitis and in other conditions.

It is important not to misinterpret the *appearance presented by the motions*. In the case of a narrow stricture low down in the rectum only fluid fæces can be passed. But with an equally narrow stricture higher up in the colon considerable solid masses may be passed. These are formed in the bowel below the stricture, are gradually accumulated, and are discharged from time to time. I have seen a fæcal mass the size of a hen's egg passed by a patient who had a stricture of the sigmoid flexure which would not admit the forefinger. The mass was a fæcal cast of the rectal ampulla. The passage of pipe-like or tape-like motions is, in the very great majority of cases, due merely to the action of the sphincter, but such motions may, however, be met with in strictures low down in the rectum. An enlarged prostate or a uterine fibroid may produce a flattened motion. When the stricture is higher up the motion may be rendered pipe-like as it passes the stenosed part, but that character must almost inevitably be lost when the matter has to travel some feet to reach the anus.

Death may be said to occur, if the general series of cases of chronic obstruction be considered, in some six months after the onset of the

symptoms of obstruction. The varieties of chronic obstruction are detailed on p. 860.

III. Chronic intestinal obstruction ending acutely.—This class of case is not uncommon. The patient has some obstruction in the bowel which does not completely block it. He has the symptoms of chronic obstruction, until upon these are suddenly engrafted the phenomena of acute or rather subacute obstruction. This sudden alteration may be due to many causes: a very narrow stricture may have become suddenly blocked by a mass of undigested food or by some foreign body. These acute attacks are very often induced by a brisk aperient, occasionally they come on after violent exertion.

In other cases the stenosed bowel has been kinked or acutely bent upon itself and so closed; or it has become the seat of a volvulus, or of an intussusception. Very often a slight attack of peritonitis—due probably to ulceration above a stricture—will bring on an acute obstruction. In not a few instances attention has been first called to a malignant stricture of the colon by an attack of subacute obstruction, the patient having previously complained only of dyspepsia, constipation and colic. A case of fæcal accumulation may end with acute symptoms if in the colon there be a definite obstructive fæcal mass.

Analysis of leading symptoms.—(*a*) *Collapse*, as an early symptom, is seen only in cases of acute obstruction. It is due to the sudden lesion inflicted upon the intestinal and peritoneal nerves. It has been described under the title of "peritonism" (see p. 616, and art "Collapse," p. 320). Its severity depends upon the suddenness of the strangulation, its rigour, and the amount and nature of the gut involved. It is more marked in connection with the small bowel than with the colon, and in the lesser bowel it is the more severe as the stomach is approached. The collapse will obviously be influenced by the age and general condition of the patient. In some cases it may be so profound as to resemble the collapse of cholera. This is especially noteworthy when the case is attended with muscular cramps (usually of the lower limbs), and possibly with some degree of diarrhœa. Later in the case the collapse is that of poisoning; the patient in acute cases, and often in chronic, dies poisoned, the septic matter entering the circulation from his own intestine. This mode of dying is practically identical with that in peritonitis.

(*b*) *Pain*, in the onset of acute cases, depends for its severity upon the suddenness of the strangulation, the amount of bowel involved, and other obvious circumstances. It certainly varies in different individuals. The first pain is due to damage to the bowel, later it is the pain of distension and of futile peristalsis, and, finally, as a rule, it is the pain of peritonitis. In acute cases the initial pain is commonly referred to the region of the navel, that is, to the region of the mesenteric and solar plexuses. Later it is possible that the pain may be definitely localised. The pain is usually a colicky pain, there is a sense of distension, or of something dragging at the bowels; there is usually an impression that if only flatus could be passed the pain would be relieved.

In the matter of diagnosis especial attention must be called to a certain feature in the character of the pain. It is this. In cases where the obstruction is complete the pain is constant, although liable to periodic exacerbations; in cases where the obstruction is but partial the pain is distinctly intermittent, and the patient has intervals between attacks of pain during which he is free from suffering. To this rule there are extremely few positive exceptions. As an illustration I may draw attention to the constant pain in acute strangulation as compared with the markedly intermittent pain in stricture. If in a case of stricture the stenosed segment becomes suddenly occluded, the nature of the pain will change almost as suddenly, and whereas before it was purely intermittent, it will become continuous. Moreover, one observes in cases of stricture that as the malady advances, and as the narrowed part becomes still more narrow, so does the pain appear at shorter intervals; until, at last, when the intestine has become entirely occluded, the pain will have become more or less continuous.

The pain in the earlier stages of intestinal obstruction is usually not aggravated by pressure. It is unassociated in fact with tenderness, and is, indeed, very often much relieved by compression of the abdomen. The appearance of tenderness is coincident with great hyperæmia of the peritoneum, or with actual peritonitis.

The diminution in the severity of the pain that is not infrequently experienced towards the end of a fatal case may depend upon the collapse following perforation, or upon diminished activity of the sensorium, or upon extensive paralysis of the intestine as a result of peritonitis, or upon a rupture or perforation of the bowel into some part other than the peritoneal cavity.

The great increase in the pain that is often experienced after food, or after the use of enemas, or even after digital examination of the rectum, depends upon increased reflex action and the fresh peristaltic movement that it excites.

(*c*) *Vomiting* is a very common symptom of intestinal obstruction. This, which appears at the very beginning of an acute case, is no doubt reflex, and is of precisely the same nature as the vomiting which may follow a wound of the abdomen or a crush of the testicle. The vomiting in the after-course of the acute case and in the chronic case depends for the most part upon the obstruction; the bowel above the obstruction becomes distended, and peristaltic movements passing along it induce a backward axial current in the contents, whereby they are poured into the stomach.

The vomiting in intestinal obstruction is characterised by its early onset, its persistence and its copiousness. At first the contents of the stomach are evacuated; then the ejected matter is bilious, and may be composed apparently of pure bile: in the next stage—if the case progress—the matter is usually thin and of a brownish colour, or it may be comparable to pea soup or be of a yellow tint like the yolk of egg.

The vomited matters soon get what is called an "intestinal odour." Finally, the matter vomited becomes stercoraceous.

The most intense and extravagant vomiting I have noted in intestinal obstruction has been in cases of plugging of the upper jejunum with a gall-stone.

Stercoraceous vomiting is more common, and appears earlier in obstruction of the lesser bowel than of the colon. In stercoraceous vomiting the matter is derived from the lower ileum or from the colon. It has been conclusively shown that for the production of fæculent vomiting it is by no means necessary to assume that matter regurgitates from the colon into the ileum through the ileo-cæcal valve. The contents of the lower ileum in normal circumstances have often the distinct characters of soft fæcal matter, and when retained in a disordered and obstructed intestine soon acquire those characters from decomposition, even if they do not naturally possess them. Nevertheless it is now fully allowed that the ileo-cæcal valve may become insufficient during life, and may permit fæcal matter to regurgitate from the colon into the lesser bowel.

This insufficiency may be met with in great distension of the cæcum and ileum associated with paralysis of the parts concerned in the valve. The occurrence, however, of this insufficiency is certainly uncommon, as is proved by repeated examinations of the parts after death from stricture of the colon.

When the obstruction occupies the lower duodenum or jejunum the vomited matters are usually very copious, and always deeply stained by bile. They can never become really stercoraceous, although if long retained they may become discoloured and acquire so offensive a smell as possibly to be mistaken for fæculent matters. In the same way the vomiting in obstruction in the middle of the ileum can never be stercoraceous in the strict sense of the term. If, however, the contents of the bowel have been long retained, as in cases where the vomiting has been subdued by opium, they may then become so altered by decomposition as to have a fæculent odour.

In some cases the stercoraceous vomiting has been due to a fistula bimucosa between the colon and the upper part of the small intestine, as occurred in a case reported by Mr. Shaw.

(*d*) *The state of the bowels.*—The constipation in cases of obstruction of the bowels depends, of course, in the main upon the narrowing or occlusion of the lumen of the intestine. It may depend also upon paralysis of a segment of the intestine without mechanical obstruction in the intestine itself, as in chronic constipation, or in cases associated with a little peritonitis. It is also to a great extent due to reflex nerve-action. Thus, in cases of acute strangulation, the constipation is often absolute from the very beginning, although the obstruction may be in the small intestine, and much fæcal matter may be lodged between the point of occlusion and the anus. Then, again, constipation is very usual in those cases of partial obstruction of the intestine when a segment of the bowel

is suddenly and severely nipped. This is well observed, as a rule, in the partial enterocele or Richter's hernia, where a part only of the circumference of the bowel is involved in the strangulation.

In cases of acute strangulation it is not infrequent for the part of the bowel below the obstruction to be emptied; and in examples where some catarrhal action has been set up in this segment of the bowel the patient may present the evidences of diarrhœa.

It is not infrequent in cases of acute obstruction for a stool to be spontaneously passed just before death. This may be derived from the bowel below the occlusion, and may be due to certain altered nerve conditions associated with impending death; or the stool may be derived from the intestine above the point of stoppage, and may indicate the yielding of the obstruction from perforation or by other spontaneous means; or the occlusion may have been incomplete, and the nerve conditions that maintained the constipation may have become modified as death approached.

In intussusception there is a sort of diarrhœa, and in a case of stricture low down in the colon this spurious diarrhœa may persist for months.

(*e*) *The state of the abdomen.*—Visible peristaltic movements and visible coils of intestine indicate a long-abiding partial mechanical obstruction, which has led to hypertrophy of the bowel above it. This feature is, therefore, of considerable diagnostic value. In advanced cases the hypertrophied coils can be seen to stand out beneath the skin. The movements of these coils is attended with rumbling and gurgling sounds and with pain. When the obstruction becomes complete the movements cease.

The meteorism in intestinal obstruction depends partly upon the completeness of the occlusion of the bowel, but more directly upon interference with the circulation through the bowel wall. This has been demonstrated by experiment in animals in whom the mesenteric veins had been ligatured, and illustrated in man by cases of phlebitis of the mesenteric veins. If the main mesenteric vein be blocked by a thrombosis the meteorism may be extreme. Advanced meteorism may exist quite independently of any intestinal obstruction. Meteorism is most marked, and is earliest seen when the colon is obstructed. In no form of intestinal obstruction is meteorism at once more sudden and more severe than in volvulus of the sigmoid flexure. In occlusions of the upper jejunum the distension of the abdomen will be confined to the region of the stomach. When the small intestines are distended and the colon empty, the median parts of the belly are protuberant. When the colon is the part distended, the coils in the regions of the cæcum, sigmoid flexure, or transverse colon are conspicuous; it is unsafe, however, to base a diagnosis as to the seat of the obstruction from the apparent situation of distended coils. Meteorism is not so much reduced by vomiting or even by diarrhœa as might be supposed. It is often reduced by strychnia, but increased by morphia.

An abdominal tumour may be felt in the following cases:—Intussusception, fæcal accumulation, cancer, certain neoplasms, and some cases of obstruction by foreign bodies.

A number of coils of small intestine, matted together by adhesions, have occasionally formed a species of tumour, and a local dulness on percussion has been caused by collapsed coils of the lesser bowel which have become grouped together below an obstruction. Such empty coils may occasionally be felt by the finger on a rectal examination, and be mistaken for a solid substance.

(*f*) *The diminution in the amount of urine* passed in many of the acute cases does not depend upon the seat of the obstruction as was once believed, but upon its acuteness, and upon the degree of the impression made upon the nervous system; it is rather one of the symptoms of collapse, and varies with the extent of the collapse and the severity of the pain. In these cases a marked increase in the amount of urine passed often attends the administration of a full dose of opium.

Obstruction in the small intestine compared with obstruction in the large.—The difference in the symptoms which depend upon the position of the obstruction are not always so well marked as is supposed. The distinguishing features by which obstruction in the small intestine can be compared with obstruction in the colon can only be given in broad outline.

Onset.—The larger number of the cases of obstruction in the small intestine are acute, while the larger number of cases of obstruction in the colon are chronic. Volvulus of the sigmoid flexure is, however, as a rule, very acute.

Pain.—In the small intestine the pain usually appears earlier, is more pronounced, more abiding, and more severe.

Vomiting.—In the small intestine, as compared with the large, this symptom appears earlier, is more distressing, and is more persistent. In the obstructions of the lesser bowel the vomited matters are often copious, are apt to be influenced by food, and more readily become stercoraceous than is the case when the stoppage is in the colon. Vomiting becomes stercoraceous on an average about the fifth day in small gut obstruction. Vomiting due to trouble in the large intestine may become irregular, may cease for a while, and may be comparatively slight; it tends to appear late and to be scanty; it is rarely fæculent until a considerable interval has elapsed.

Constitutional disturbance is, other things being equal, more marked in small gut obstructions than in those of the colon. There is in the latter a greater tendency to severe collapse, and consequently a more frequent appearance of the various phenomena indicative of shock.

Meteorism.—When the lower part of the small intestine is obstructed, the meteorism may first show itself, and remain for a while most marked, in the hypogastric, epigastric and umbilical regions. In well-marked cases the abdomen presents the appearance of a six months' pregnancy, and the flanks and iliac fossæ are depressed. This symptom, however, is of no

great value, for the appearance may be imitated almost exactly by a distension of the sigmoid flexure, when that part of the gut forms a large coil which projects towards the middle line of the abdomen.

When the lower part of the colon is involved, the meteorism will attain a much greater degree than it does in small gut obstructions. Indeed, upon the whole, it may be said that meteorism is less in the latter cases than in the former. Distension of the colon, especially with solid matters, may map out very precisely the anatomical outline of the bowel, and leave the central parts of the abdomen comparatively undistended. In any case, however, the meteorism tends to become general, and any appearances that may be regarded as characteristic are soon lost.

In diagnosing acute volvulus of the sigmoid flexure from some acute strangulation of the small intestine, great importance attaches to the rapid development of a high grade of meteorism in the former affection.

Apart from this point in diagnosis, I do not think that anything like the importance attaches to meteorism as a means of diagnosis that has been ascribed to it. Speaking in very general terms, it may be said that meteorism in connection with obstruction in the lesser bowel, as compared with that in the colon, appears comparatively late and is seldom of high grade.

Enemas.—A great deal has been written by various authors upon the value of enemas as a means of diagnosing the seat of the obstruction. The test consists in a comparative estimation of the amount of water that can be held by certain segments of the bowel. Thus elaborate statements have been made to the effect that if a certain amount of water can be readily injected, then the obstruction must be in the sigmoid flexure; if a certain additional quantity can be introduced, then the stoppage must be in the descending colon; and, finally, if a certain number of pints can be received, then the whole of the large intestine must be occupied and the occlusion must be situated in the small bowel. The statements are usually based upon experiments on the capacity of the various segments of the colon made upon the cadaver.

For diagnostic purposes this method is, I venture to think, absolutely useless. In the first place, observations made upon the cadaver, where the parts are relaxed and where muscular action has ceased, do not agree with those made upon the living subject. The method, moreover, does not take into consideration the condition of the bowel below the obstruction. This part of the tube may be dilated or contracted, may respond vigorously to certain forms of irritation, or remain absolutely inert. Once more, as Hilton Fagge pointed out, there are certain strictures, especially those associated with some bending of the gut, or with a valvular arrangement of the mucous membrane above the stenosed part, through which water may be injected from below, while fluids above the stricture are unable to escape.

The passage of the long tube.—In this method a flexible tube or sound is passed into the rectum, and an attempt is made to diagnose the seat of the obstruction by noting the distance to which the tube can be in-

troduced. This procedure applies mainly to stenosis of the lower part of the colon; but as a means of diagnosis it is entirely valueless. In some cases the sound lodges early in its course against a fold of mucous membrane, and the diagnosis of an obstruction low down in the bowel is made accordingly. In other instances in stricture of the end of the sigmoid flexure, the tube may reach the upper extremity of the rectum, and then turn upon itself, or become coiled up in the rectal ampulla, until so much of it has been introduced that the whole of the colon from the splenic flexure downwards has been pronounced free from obstruction. I have good reasons for doubting whether these rectal sounds ever go beyond the sigmoid flexure, reasons which are fully confirmed by experiments I have made upon the dead body. If the segment of the colon which forms the sigmoid flexure and the free part of the rectum be uncoiled, it will appear in the form of a large loop of intestine extending from the psoas muscle to the spot where the rectum becomes fixed about opposite the middle of the sacrum. This loop has the outline of a capital omega, and is usually provided with an extensive mesocolon. Occasionally this mesocolon is so long that the summit of the omega loop can be made to touch the cæcum or to reach the level of the umbilicus. In some examples I have found this loop to be from eighteen to twenty inches in length. If the long tube be introduced into such a coil, its extremity may reach beyond the level of the umbilicus, and yet not have passed beyond the sigmoid flexure.

Differential diagnosis of intestinal obstruction.—If the case be (A) **acute** it may most probably be placed under one of the following headings:—(*a*) Strangulation by bands or through apertures, including strangulation by adherent appendix and the like. (*b*) Volvulus of the sigmoid flexure: other forms of volvulus are rare. (On page 815 the clinical features of the less common forms of volvulus have been already dealt with.) (*c*) Acute intussusception. (*d*) Acute obstruction by gallstones or foreign bodies. Under these four headings the common forms or types of acute obstruction may be arranged. Anomalous cases are represented by unusual forms of obstruction such as are described on page 808. The clinical phenomena of these comparatively rare varieties have been already considered.

B. If the symptoms be **chronic**, the trouble may be due to (*a*) Stricture of the same intestine, including the many conditions in which the lesser bowel has its lumen partly occluded by adhesions, by matting together, by compression, by bending, by obstructing substances, or by new growths; (*b*) Stricture of the colon, including the many conditions in which the colon has its lumen partly occluded by compression, by adhesions, by bending, by obstructing substances, or new growths; (*c*) Fæcal accumulations; (*d*) Chronic intussusception.

C. The obstruction may have been **chronic** in its general course, and may then **terminate acutely**.

The cases which would come under this heading have been already considered (page 839); they are cases of chronic obstruction in which the

symptoms of that variety are present, but in which the already narrow bowel becomes occluded suddenly and acute symptoms are produc This variety is best illustrated by a stricture of the colon, in which t narrowed part of the gut becomes suddenly occluded by bending kinking of the bowel, or by the blocking of its lumen by a foreign bo which has been swallowed, or by a mass of undigested food. Simil acute phenomena may supervene in any case of chronic obstruction.

A. The acute cases.—The following are the *general phenomena* acute intestinal obstruction.

The attack, in over 80 per cent of the cases, is sudden in onset. T patient is seized with acute *pain in the abdomen.* This pain may be ve intense, may cause the patient to be "doubled up," or even to roll the floor; it is relieved by pressure. The pain is somewhat like th of colic. It is often described as a "tearing" pain. It is continuo but with exacerbations. The patient often feels that if he could pa flatus or a motion the pain would be relieved. With this violent pa is associated more or less collapse, which may be profound, and m imitate the collapse of cholera. In almost all cases there is profou exhaustion with pallor and faintness, and an expression of intense anxie and distress.

In the majority of cases the pain is referred to the immediate vicini of the umbilicus. The instances are few in which it may be relied up to indicate the seat of the obstruction. The pain at the umbilicus usually, no doubt, a referred pain, the site being the mesenteric plex The localisation of the pain is often very misleading, as the followi examples will show:—The pain was on the right side just below the live the obstruction was in the ileum, 18 inches from the cæcum.—The pa was on the left side, and on a level with the navel, and in one ca where it was so placed a coil of ileum had passed through a rent in t right broad ligament, while in another the strangulation was deep in t right iliac fossa.—The pain was near the gall-bladder; the obstructi was in the ileum.—The pain was in the epigastrium, and the troub which caused it was due to a band passing between the urinary bladd and the lumbar spine.

It may be said, then, that the position of the pain in acute intern strangulation is of no use as a guide to the seat of the lesion, but is rath misleading; and that it is more often complained of about the nav than elsewhere.

The pain that is so conspicuous a feature at the beginning of the cases persists throughout the course of them. It does not, howeve retain its original intensity; it soon becomes less severe, and oft undergoes considerable abatement. In some of the more acute cas however, it has persisted with all its original intensity until deaden by collapse.

The pain often ceases shortly before death; a relief, however, of hopeful significance: it is usually coincident with a profounder collap

with gangrene of the bowel involved, or with advanced narcotism or septicæmia.

There is more or less direct connection between the intensity of the pain and the severity of the other symptoms. The attack may set in during sleep, or follow severe indigestion or constipation, or attend some violent exertion. It has appeared to be induced by a purgative. In the majority of instances, however, no exciting cause is apparent, the patient being in good health, or suffering at the most from some abdominal uneasiness.

Local tenderness, as demonstrated by pressure upon the abdomen, is, as a rule, entirely absent at first, when indeed the patient may be relieved by pressure upon the belly walls. Tenderness may never appear at all, especially in cases pursuing a rapid course. In a few cases of less acute character it has been trifling, or not sufficiently marked to attract notice. In the majority of cases, however, some part of the abdomen becomes tender during the course of the disease. This tenderness may be limited in extent, or diffused. Limited tenderness usually appears about the second or third day; it is a symptom that, when well marked, is of some diagnostic value, since it appears to be restricted to the actual seat of the lesion. It depends, no doubt, upon congestion or inflammation of the involved coils, or upon some peritonitis excited in their serous coat. As a factor in diagnosis, then, it is of more value than the spontaneous pain observed in these maladies.

A diffused and well-marked tenderness generally indicates the onset of a peritonitis, and is also a symptom of clinical value. When peritoneal inflammation has become diffused a general tenderness is practically constant, unless modified or concealed by profound collapse or narcotism.

Vomiting is a conspicuous and constant symptom. In a few cases it has been the earliest symptom of the obstruction; in the great majority it comes on immediately after the appearance of the pain, or within a few hours after that event. I have met with instances where the vomiting did not appear until twenty-four hours after the onset of the pain.

The ejected material consists of the contents of the stomach, and then usually of bilious matters. In its next stage it may be thin and of a brownish colour, or be comparable to pea soup, or be of a yellow tint. Vomited matters with these characters are often described as possessing an "intestinal odour." Lastly, the vomit may become stercoraceous.

Stercoraceous vomit is common in this form of obstruction, and occurs, indeed, in between 60 and 70 per cent of the examples. The period in the attack at which the vomit assumed a fæculent character varied from the second to the ninth day. An average taken from a large number of cases gives the fifth day as the mean. The cases in which the vomited matter does not become stercoraceous are represented by those in which death occurs at an early period, or by those in which the progress of the case is less acute than usual.

The vomited matter very rarely contains blood.

When once it has set in, the vomiting will persist until the end of the attack. It is one of the most distressing of the symptoms. Every-thing swallowed is immediately ejected, and even when nothing is taken by the mouth the vomiting will continue incessantly. Often a little movement or a little pressure upon the abdomen will excite an attack. When not actually sick the patient will commonly complain of a most distressing nausea, and will be troubled by eructations of flatus It is worthy of note that the patient is in no way relieved by the attack of vomiting, as may be the case in other maladies associated with this symptom. With few exceptions, the longer the obstruction lasts the more violent and distressing do the attacks of vomiting become. Sometimes they may cease entirely a few hours before death, just as under the same circumstances the pain may abate. In other cases, however, there has been a sudden and profuse gush of vomit either just before death or in the act of dying, the fluid pouring, without effort, from the mouth and through the nostrils. This symptom is sometimes observed also in death from peritonitis. In a few cases, where the obstruction does not appear to have been very complete at first, the vomiting distinctly abated after the violent attack marking the onset of the trouble had passed away.

Opium has often a very decided effect upon the vomiting. When the patient is well under the influence of the drug the symptoms of intestinal obstruction may be more or less efficiently masked. The pain abates, the pulse improves, the amount of urine, if lessened, increases and the vomiting becomes less troublesome or ceases for a while. Under the influence of opium even stercoraceous vomiting may cease, and on the reappearance of the symptom the ejected matters may be not fæculent.

Peritonitis, presumably by the paralysing effect it has upon the intestine, seems to have some influence upon the production of fæculent vomiting. When acute peritonitis sets in early the tendency for the ejected matter to become stercoraceous is certainly much less. In some cases this change has been very marked.

In nearly every instance the act of vomiting is associated with much retching and distress.

Constipation, as a rule, is marked from the first, and is absolute neither fæces nor flatus being passed.

As a rare circumstance a motion may be passed during or immediately after the occurrence of the initial symptoms; it comes from the bowel below the obstruction. Enemas administered almost at any time after the onset of the attack may possibly bring away fæcal matter from the colon. Flatus generated in the large intestine may also be passed but the circumstance is quite exceptional.

I have met with two or three recorded instances where blood is said to have been passed in acute cases other than intussusception.

A more or less copious motion may be passed just before death or the act of dying.

General constitutional symptoms.—An initial rigor is exceedingly rare

Collapse is one of the earliest symptoms, and may be profound or even fatal. There is great muscular weakness. The face is drawn with pain and has an aspect of anxiety; the features become pinched, the eyes sunken, the voice weak and muffled, and a cold sweat covers the surface. In those extreme cases which may be mistaken for cholera, the limbs become cyanosed and the complexion livid. The patient usually dies with those manifestations of general septic poisoning which mark the termination of fatal peritonitis (see p. 613). The intelligence, as a rule, is retained to the last. The pulse is small, rapid, and thready, and varies with the general condition; it is usually improved up to a certain point by opium.

The temperature, as a rule, is subnormal throughout. Generally speaking, the onset of peritonitis does not affect it, but now and then there may be a feeble reaction, and the temperature may reach to 99° or 100°. Such examples are not common. On perforation the temperature is that of profound collapse.

The respirations are superficial and thoracic. Should the abdomen become much distended the breathing may be much embarrassed.

The tongue is usually coated, being at first white and then dry and brown. The mouth is parched, and a very offensive taste is complained of.

Intense thirst is usually a marked and distressing symptom. It is most marked in instances associated with profuse vomiting, and in those attended by peritonitis.

Obstinate and distressing hiccough is occasionally a prominent feature. It is rare.

The quantity of urine is very commonly diminished, and in the most acute cases may be entirely suppressed, the bladder being found empty. As has been noted, the effect of internal strangulation upon the renal excretion is brought about mainly through the nervous system. A diminution in the amount of the urine is most marked, therefore, in the most acute cases, and in those attended by intense pain and much collapse. In many instances the excretion of the urine has been immediately increased on the patient coming under the influence of opium. The position of the obstruction in the small intestine has no effect upon this symptom. It may be absent when the strangulation concerns the jejunum, and present when it involves the ileum.

Strangury is very rarely noticed. In one instance of this symptom the obstructing band was attached to the bladder. In another so large a mass of empty coils hung down into the pelvis that it may possibly have pressed upon the bladder. The patient was a girl aged ten, and the mass was found, during life, to press upon the rectum.

Tenesmus is practically unknown in the acute cases, with the notable exception of intussusception.

In some 6 per cent of the cases of acute strangulation cramps are complained of. The subject of muscular spasm in connection with strangulation of the bowel has been fully investigated by M. Berger. He finds that the cramping pains are usually in the feet and calves, that

the symptom is limited to cases of severe strangulation, and is most common in adults. He has collected fourteen cases where this feature was noted. Eleven were cases of strangulated hernia, two of strangulation by a band, and one of obstruction by a diverticle.

It is in a case of this kind, attended with cramps in the limbs, and profound collapse, with a cold skin and cyanosed extremities, that the mistake of diagnosing intestinal obstruction for cholera has occurred. This error may well be excused when the strangulation has been preceded by an attack of diarrhœa.

The *abdominal walls* in most instances remain flaccid, or in their normal condition, until such time as local or general peritonitis sets in, or distension reaches a considerable degree. Even in some cases where peritonitis was found after death the parietes have retained their normal suppleness to the end.

Meteorism.—Distension of the abdomen in most varieties of this form of obstruction is comparatively slight. It usually appears about the third day. It appears to be least marked in the rapid cases, and especially so in cases attended by extreme vomiting. When peritonitis sets in, the meteorism undergoes a considerable increase. The swelling is usually noticed first in the epigastric and umbilical regions, and may form a very distinct elevation of the parietes in those districts. A special exception must be made of volvulus of the sigmoid flexure, in which meteorism is early, is pronounced, and is, at first, often localised.

The question of any tumour or area of limited dulness within the abdomen is discussed under the respective headings.

(*a*) *Strangulation by bands or through apertures.*—Under this heading are included the following: — Strangulation by isolated peritoneal adhesions; by omental cords; by Meckel's diverticulum; by normal structures abnormally attached, as by an adherent appendix or Fallopian tube: strangulation through slits and apertures of various kinds.

The instances of obstruction that come under this heading form collectively more than one-fourth of all the varieties of intestinal obstruction.

Certain anomalous forms of obstruction due to isolated bands and adhesions are dealt with on page 808.

The clinical phenomena in these cases are identical with those just described as characteristic of acute intestinal obstruction. The leading features are, indeed, those of an acutely strangulated hernia. They may be summarised as follows:—

History.—The patients are mostly young adults. In 68 per cent of the cases there is a history of previous peritonitis. The onset is sudden.

Pain appears early, is very severe and persistent, and is mostly located about the navel.

Vomiting appears early, is a marked symptom; constant, copious, and severe. In 60 per cent of the cases it becomes stercoraceous, on an average on the fifth day. It affords the patient no relief.

Constipation is continuous and absolute from the first. Enemas may

evacuate the contents of the colon. There is no discharge of blood from the rectum.

Prostration is marked; there is often profound collapse, intense thirst, diminished urine, etc.

Tenesmus is absent.

The abdominal parietes are flaccid, unless peritonitis has set in.

Meteorism is slight. It appears usually about the third day, and in most cases involves first the epigastric and umbilical regions. Local tenderness of the abdomen is absent, at any rate at the outset.

Tumours or localised districts of dulness caused by the distended and strangled loops are extremely rare, and are, in any case, very indefinite.

Coils of intestine are not visible through the abdominal walls.

The average duration of this variety is about five days. In the very acute cases death may take place within twenty-four hours. In the less acute cases patients have lived twelve and fifteen days.

(*b*) *Volvulus of the sigmoid flexure.*—This is the only common form of volvulus. Other varieties of volvulus are dealt with on pages 817, 818.

Sex; Age.—Volvulus of the sigmoid flexure is more common in men than women, in the proportion of four to one. It is very rare before forty; the patients' ages are usually between forty and sixty.

Previous history.—In nearly every case there is a history of previous constipation. Some particularly indigestible food may have been swallowed. There is often a history of colic relieved by placing the body in a certain posture.

The mode of onset is usually sudden.

Pain appears early, is a marked symptom, is severe but not usually so severe as in the previous form, is commonly intermittent at first becoming subsequently continuous but with exacerbations, is very often complained of about the umbilicus, and later about the region of the sigmoid flexure.

Local tenderness appears early over the region of the distended coil of colon, and is constant.

Vomiting appears less early, and is less marked and severe than in the previous form of obstruction. It may be absent. It is often scanty. It is stercoraceous in only 15 per cent of all the cases. It may abate. It often affords much relief to the patient.

Frequent eructations are very common.

Constipation is early and absolute. There is no discharge of blood from the rectum.

Prostration is not so marked as in the above form of strangulation. There may, however, be marked collapse with diminished urine. Intense thirst is not a marked feature as a rule. The patient often suffers from dyspnœa and a sense of suffocation, symptoms not met with in the previous class of cases.

Tenesmus is a marked feature in 15 per cent of the examples.

The abdominal walls soon become rigid, owing to the early and almost constant appearance of peritonitis, general or local.

Meteorism appears very early, increases rapidly, and becomes extreme. The thoracic viscera are often displaced by the distended intestines.

Tumours of a definite character are not met with; nor are coils of intestine visible.

The mean duration is six days. In one case, at least, the patient died within sixty-four hours. Another patient lived twenty days.

(*c*) *Acute intussusception.*—Sex; Age.—Acute intussusception is somewhat more common in males. It occurs mostly in the young, and more than 50 per cent of all cases occur under the age of ten years; 25 per cent occur during the first year of life. After forty the trouble becomes rare.

Previous history.—There may be nothing noteworthy in the history. Often the child has eaten some indigestible food, such as nuts. Exposure to cold, effort, and violence have all been noticed occasionally. In a few rare instances there is an account of previous attacks of obstruction, due probably to intussusception.

The mode of onset.—This is sudden in 75 per cent of the cases. The symptoms may appear during perfect health, and may come on during sleep. Very often there is a preliminary colic, or some diarrhœa, or bowel disturbance.

Pain.—This is one of the first symptoms. Often severe at first, it increases up to a certain point, and then subsides. It is not so severe, on the whole, as in strangulation by bands. The pain in any given case may begin gradually in the form of trifling attacks of colic, appearing at long intervals or coming on only after defæcation; or a violent initial attack may be preceded by a definite but trifling sense of discomfort in the abdomen. The form of invagination that is most usually associated with intense pain at the onset is the ileo-colic.

The pain is colicky, and its great feature is its occurrence in paroxysms. Intermittent pain, as has been already stated, nearly always indicates an incomplete obstruction in the intestine, and in intussusception it may therefore be expected to be well marked. The pain may at first occur at long intervals, during which the patient is free from suffering; but as the malady advances the intervals become shorter and shorter. In the acuter forms the intervals are not marked. The patient very often is never free from pain; but here, although the pain is continuous, it is broken in upon by definite exacerbations. The intervals between the attacks are sometimes very precise, the paroxysms appearing every twenty or thirty minutes, and having a more or less exact duration. In any case, as the intussusceptum becomes congested, its neck more and more strangulated, and its lumen narrowed, the pain becomes more continuous; although it is still associated with exacerbations. When the paroxysms are marked they usually appear suddenly and subside suddenly, although to this course there are many exceptions.

The pain in intussusception depends upon violent and irregular peristaltic movement. It is more severe, as a rule, in cases involving the

small than in those involving the large intestine. Some of the most severe instances of pain have been in the ileo-colic varieties, and in invaginations high up in the small intestine where the muscular coat is well developed. It has been said that the intervals between the paroxysms are shorter when the small gut is involved, as compared with the colon. This is often true, but the fact depends rather upon the greater degree of occlusion met with in the lesser bowel than upon the anatomical position of the lesion. Everything depends upon the state of the intussusception itself; a small invagination in the colon may cause early and intense pain, while, on the other hand, an ileo-cæcal invagination may actually project at the anus before much pain has been produced.

I cannot support the statement that the more empty the bowel the less the pain.

In position the pain is at first very ill-defined, but as the invagination advances, and especially as a definite tumour is formed, it becomes more or less distinctly localised about the seat of the lesion.

At first the abdominal walls are not tender on pressure; it often happens, indeed, that pressure over the more painful part relieves the patient's suffering. In intussusception the abdomen in time usually becomes somewhat tender on pressure, especially about the site of the invagination. This is partly the result of continued irregular muscular action, but is perhaps to a greater extent due to the engorgement of the invaginated parts and to the appearance of some local peritonitis. In the absence of a definite tumour a definitely-localised tenderness is a valuable guide to the position of an intussusception. Sometimes the pain is relieved when the patient assumes a peculiar posture. The longer the case lasts the greater is the tendency for both the pain and the tenderness to become diffused, presuming that they have been previously localised.

Vomiting in intussusception is by no means so conspicuous a symptom as it is in other forms of acute intestinal obstruction, such as in strangulation by bands. It does not appear so early; it seldom becomes excessive or very distressing; it is less often stercoraceous, and is apt to fluctuate considerably.

In about three-fourths of the acuter cases it appears with the earliest symptoms, coming on either with the pain or a little while after it. In the remaining cases it appears later, and on an average about the third day. In about 8 per cent of the acute and subacute cases vomiting does not appear to have occurred at all during the course of the malady.

There is often great irregularity in the appearance and character of the sickness. Indeed, as a rule in intussusception this symptom is marked by considerable fluctuations. In many cases the vomiting, after having been severe, has been absent for several days together. In several examples of the acute form of the malady which I have collected the patient was only sick once, while in other instances the vomiting appeared at long and irregular intervals. The attacks of vomiting often coincide with attacks of pain. In one case of acute invagination where

the sickness had ceased the symptom was set up afresh by introducing the finger into the rectum.

The examination of a number of recorded cases shows that the vomiting is least severe and most inconstant in those cases that are associated throughout with diarrhœa. Very often also it is slight in degree in those instances of the malady that are attended by distinctly paroxysmal pain. In other words, the sickness is least troublesome when the lumen of the bowel is still patent. Most of the worst instances have been in cases marked by early and persistent constipation, excluding from that term the passage of blood and mucus unmixed with fæces. In any case the sudden cessation of diarrhœa is usually attended by an increase in the vomiting.

In many instances the vomiting gives much temporary relief. This is especially the case when it appears at long intervals. This feature is more marked in the vomiting of intussusception than in any other form of obstruction.

The vomited matter is usually alimentary or bilious. Fæculent vomiting is not met with in more than 25 per cent of all cases of acute or subacute intussusception. In chronic cases it occurs only in about 7 per cent. In the acuter cases stercoraceous vomiting is in nearly every instance associated with constipation, or at least with the passage of no fæcal matter in the discharge from the anus. It is met with most frequently in invaginations about the ileo-cæcal region, and then in those involving the lower extremity of the small intestine. It appears, on an average, on the fourth or fifth day. Often, however, it does not appear for a week or a fortnight, or not until near the end of the case, when the progress of the malady is distinctly subacute. In two or three instances blood has appeared in the vomited matter. This symptom is usually met with in children and in enteric intussusceptions.

On the whole, it may be said that vomiting is most marked in the enteric and ileo-colic invaginations, less marked in the ileo-cæcal forms, and least so in the colic and rectal varieties.

The state of the bowels in intussusception presents very distinct characters. As a result of the violent peristaltic action excited by the invagination, diarrhœa is a very common condition; and as a consequence of the great engorgement of the intussusceptum it follows that the motions passed are usually stained with blood. When the lumen of the bowel becomes so occluded that no more fæcal matter passes, the evacuations may consist simply of bloody mucus.

Constipation, as indicated by the passage of no fæcal matter, is not common in intussusception. In the majority of the acute and subacute cases there is some diarrhœa at first, and then absolute constipation towards the end of the case. The occurrence of more or less constipation as a marked feature during the progress of the malady does not pertain to more than 30 per cent of the cases. Sometimes diarrhœa continues throughout the whole course of the case, being, as a rule, more marked at the beginning than at the end. At the same

time it may be noticed that a severer diarrhœa, or a diarrhœa after constipation, may precede, attend, or follow the elimination of a gangrenous intussusceptum. Sometimes a loose state of the bowels alternates with some constipation, but this condition is more usual in the chronic forms of the malady. The diarrhœa may be severe; and from ten to twenty evacuations may pass in the twenty-four hours.

The occurrence of blood in the stools is a striking feature. As a rule, the more acute the case and the more violent the strangulation, the more conspicuous the hæmorrhage. In acute cases this symptom is present in about 80 per cent of the examples. It is met with less frequently in those following a subacute course, and is found in no more than 50 per cent of the chronic cases. It is perhaps more marked in children than in adults. It is most frequent in the ileo-colic varieties, then in the ileo-cæcal, next in the colic, and is probably least frequent in enteric invaginations. The amount of blood is usually not excessive; the hæmorrhage may, however, be so profuse as to be the principal cause of death; the clots of blood may block up the lumen of the intussusceptum, and may even plug the bowel below the seat of the invagination. In any case the symptom is usually more marked at the outset of the attack than during its later progress. Bleeding may, however, attend the evacuation of the intussusceptum.

Tenesmus is a striking symptom. It is more commonly met with in acute and subacute than in chronic cases. Indeed, other things being equal, the more chronic the case the less frequent is the appearance of the symptom. I find that in acute and subacute forms tenesmus occurs in about 55 per cent of the examples. Rafinesque finds an account of the occurrence of this symptom in only 13 per cent of distinctly chronic cases. The frequency and severity of the tenesmus depend mainly upon the nearness of the intussusception to the anus. The symptom, therefore, is very usual in rectal and colic invaginations, is common in the more extensive ileo-cæcal varieties, and is least often met with in the pure enteric forms.

Tenesmus is usually an early symptom of intussusception, and indeed is often among its first manifestations. It may be so persistent and so severe as to cause intense distress. When the invagination occupies the rectum or sigmoid flexure the tenesmus may be followed by paralysis of the sphincter ani, whereby a patulous condition of the anus is produced.

Prostration.—In the more acute cases there is much collapse, especially in the young; but, on the whole, the amount of shock is less than in strangulation by bands. In the acute cases the temperature is subnormal. In the majority of the cases, and especially in those distinctly subacute, the temperature is normal or a little above subnormal.

Intense thirst, as in cases of strangulation by bands, is not common.

The child is very restless and peevish.

The abdominal walls are flaccid unless some peritonitis has set in.

Meteorism is quite rare except in cases associated with pronounced

constipation. It is thus most commonly met with towards the end o the attack. When diarrhœa exists, not only is no meteorism present, but, on the contrary, the abdomen is often distinctly sunken. On the cessation of the diarrhœa the symptom may appear. It is usually quite moderate in degree. It is needless to say that it appears to a greater or less extent when peritonitis sets in.

The "signe de Dance" is of little or no value. It is said to occur in cases where the cæcum has become invaginated, as in the ileo-cæca forms of the disease; it consists in a depression about the right flank o right iliac fossa. It is supposed to indicate the displacement of th caput coli.

A tumour.—The presence of a tumour formed by the invaginate mass, and to be felt either through the abdominal parietes or rectum, i to be discovered in a little less than 50 per cent of all cases, and woul appear to be not more frequently felt in the chronic than in acu forms. The tumour is most frequently associated with the ileo-cæc and the colic varieties, least frequently with the enteric and ileo-coli It is usually more distinct in children than in adults. The tumo varies in size. It may be as small as a hen's egg, or it may attain th thickness of the adult forearm. It is cylindrical, and is very common described as sausage-shaped. It often shows the distinct curve of th intussusception. As regards length, it is usually short, and very rare exceeds six inches. This limitation in length does not necessari correspond to the length of the invagination mass; it depends rath upon its position. The tumour is not evident when it occupies t hepatic or splenic flexures of the colon, and thus the portion that can detected cannot well exceed the length of the transverse or descendi colon, or of part of the right limb of the large bowel.

Sometimes it assumes the appearance of a double tumour, one pa having been felt in the transverse and the other in the descending colo the intermediate portion in the splenic flexure not being evident. the ileo-cæcal variety the tumour will be more distinct the nearer t mass to the rectum; while in the cæcum and lower ascending colon t tumour must necessarily be small. The rarity of a tumour in the ile colic variety is explained by the small size of those invaginations wh simple, and by the fact that the intussusceptum is composed of sma intestine enclosed in large. Thus it happens that the tumour is most oft met with over the descending colon, and next in frequency over t transverse colon. Enteric invaginations usually form a tumour in t cæcal region, the lower ileum being the part most often involved.

The tumour varies in distinctness, and it is seldom that all parts of can be made out equally well. It usually appears fixed; but it m often, especially in chronic cases, be observed to change its positio now to advance along the colon in the direction of the anus, and now return in the inverse direction. It can often be made to move under t use of an enema, the mass being forced back towards the cæcum; th can only occur in invaginations that involve the colon. The progress

the invagination from the cæcum to the rectum can often be distinctly watched. A tumour which remains long stationary in the cæcal region probably depends upon an ileo-colic invagination.

In consistence it feels hard and resisting. Its density may vary greatly. During attacks of pain it may be large, prominent, and hard; during the intervals it often becomes less distinct and softer. When first noticed it frequently happens that it is only present while painful peristaltic movements are going on, being quite absent when the patient is free from pain. When it has existed for some time it is generally tender; but in earlier periods any pain that may be felt in it is often relieved by pressure. In a doubtful case an examination of the abdomen should be made under chloroform.

In about 25 per cent of the cases in which a tumour is present it may be felt in the rectum or may project at the anus. This condition, as may be surmised, is almost limited to the colic and ileo-cæcal invaginations. It appears much more frequently in children than in adults; in children, moreover, the tumour reaches the rectum much more quickly, owing to the greater mobility of a child's colon. In such patients it has reached the rectal region by the second day of the attack, and, as already stated, may be one of the early evidences of the invagination. As a rule, the tumour appears in the rectum much later; on an average (in acute and subacute cases) on the seventh day. In chronic forms the average date for the appearance of the mass in the rectum is the fifteenth day. It has, however, appeared as late as the third and fourth months, and in one case as late as the seventh month of the duration of the symptoms. The protrusion is usually small (being about three inches in length) and conical in shape; but it may attain greater length (I have seen one eight inches long), and cases are reported where ten and twelve inches of bowel have projected from the anus. The protruding mass is usually deeply congested and much altered in structure. It may be gangrenous. Even when it has protruded for some time at the anus the intussusception has been successfully reduced by enemas, insufflation, or laparotomy. At the apex of the projecting tumour the ileo-cæcal valve may be presented, and near its extremity the orifice of the appendix. When examined by the finger introduced into the rectum, the tumour, before it has prolapsed, presents tolerably characteristic features to the touch; its swollen extremity with its narrowed lumen has been many times compared to the os uteri.

The tumour when in the rectum, or when protruding beyond it, has often been the cause of an error in diagnosis; it has been mistaken for prolapse, for rectal polypi, and for piles. Unfortunately the error has sometimes extended from diagnosis to treatment, and the mass has been incised, or cauterised, or even cut off.

(*d*) *Acute obstruction by gall-stones, foreign bodies, etc.*—There may be a history of foreign bodies swallowed. Gall-stones are much more common in women than in men. The average age is fifty to sixty-five. Gall-stones may have been passed. There may be no history of hepatic colic. Stones which cause obstruction have passed direct from

the gall-bladder to the duodenum or neighbouring small intestine b ulceration; a stone which could pass the common bile-duct could not d itself occlude the bowel.

It will be convenient to deal with obstruction by gall-stones as typica of the present series of cases.

In the first place, if there need be no history of hepatic colic there is often evidence, on the other hand, of a previous local peritoniti in the neighbourhood of the gall-bladder, such "attack" representing th time of the passage of the stone from the gall-bladder. In man instances symptoms of obstruction have appeared very soon after th supposed entrance of the stone into the bowel; in other cases a lon interval has elapsed.

In perhaps a large number of cases a gall-stone of considerabl magnitude may pass along the bowel without exciting any marked di turbance, and may only cause trouble when it comes to be evacuated the anus. In other instances the passage of the calculus is marked b attacks of colic from time to time, by irregularity in the motions, b some meteorism, and by vomiting. The symptoms may be severe whi they last, and indicate, no doubt, a complete but temporary obstructio The symptoms after being violent are not infrequently relieved suddenl and the patient passes in a few moments from a state of intense sufferi to a condition of almost perfect ease. Such a transition is probably coi cident with the passage of the concretion through the ileo-cæcal valve in the colon, where it ceases to give trouble. The length of time which m intervene between an attack of obstruction and the actual passage of t stone may be considerable; it may amount to one or two weeks.

In a great number of cases the clinical history is as follows:—T patient dies of an attack of acute intestinal obstruction. In the attacks there is pain more or less persistent and of a colicl character, vomiting that is usually very copious and may become ster raceous, constipation that soon passes into absolute obstruction a more or less meteorism of the abdomen. These attacks, as a rule, do n arise suddenly. They are not associated with the severe pain th marks some forms of acute obstruction, such as strangulation by a ban and, as a consequence, they are not attended by so much collapse. T vomiting is often very frequent and copious, and in the majority of cas where the lower ileum is concerned, becomes fæculent before deat There is no marked abdominal tenderness unless peritonitis be setting i It is only in the rarest instances that any tumour has been detected.

So far as recorded cases show, the average duration of these acu final attacks is seven days, the shortest period being four days, t longest fifteen days.

These attacks have often been preceded by similar evidences obstruction, which may or may not have been brought about by t same stone. Thus one patient had two attacks, the previous o occurring three months before death; another had three attacks whi appeared eighteen months, twelve months, and six days respective

before death. In another instance the patient is described as having had many attacks like that which proved fatal in the end.

In the intervals between such attacks the bowels have usually been irregular and the patient liable to digestive disturbances and to sickness.

In another set of cases the obstruction leading to death has been more chronic. There has been, perhaps, absolute constipation for twenty days before the decease of the patient, and the progress of the case may have been indolent and gradual. Such cases also may or may not have been associated with previous attacks of intestinal disturbance. In these more chronic cases all the symptoms are less marked; the pain may be intermittent, the vomiting is less pronounced and is rarely fæculent, there may be some meteorism, and the coils of intestine may be visible through the parietes.

In a third series of cases the stone would appear to cause but partial obstruction, and symptoms are produced which are identical with those of stricture of the small intestine.

Concerning partial obstruction, it should be noted that an impacted calculus may in time push out a diverticulum from the intestinal wall, and thus become encysted without offering a great obstacle to the passage of intestinal matters. This is said to occur most frequently in the duodenum, but probably the cases thus described are rather instances of a gall-stone making its way towards the duodenum by direct ulceration from the gall-bladder.

It will be obvious that the symptoms must depend somewhat upon the position of the stone in the intestine. The nearer the obstruction to the stomach the more marked is the vomiting, and the less marked or the longer delayed are the evidences of interference with the action of the bowels. In some cases of impaction in the jejunum the vomiting has been very severe and remarkably copious. The symptoms have become almost immediately exaggerated by the taking of food, while constipation has not become pronounced until the other symptoms have existed for some time. In a case of Dr. Pye-Smith's, in which the gall-stone was in the upper part of the jejunum, the vomiting was profuse; no less than one and a quarter gallons of bilious fluid were ejected in forty-eight hours. The patient lived six days.

Obstruction is often assisted by abrupt bending of the bowel at the point of impaction of the stone.

Spontaneous evacuation of the stone may occur even after symptoms of great severity. Thus, in the case recorded by Dr. C. Martin, the patient suffered from absolute obstruction lasting six days; the vomiting became severe, and was at last stercoraceous, but on the morning of the seventh day a motion was passed which was followed by the evacuation of a large stone. The patient rapidly recovered. The concretion had a circumference of 3½ inches.

In another case, quoted by Dr. Sands, a woman, aged forty, suffered from obstruction due to the impaction of a gall-stone. The constipation was complete for four weeks. At the end of that time a motion was

passed, and was followed seven days later by a biliary calculus wit circumference of three inches. Stercoraceous vomiting set in on t third day and lasted for three weeks. The patient had been treated aperients and by enemas. She made a good recovery.

The symptoms produced by *enteroliths*, which may be convenien considered here, vary greatly, and depend a good deal upon the situati of the mass in the intestine. They very rarely cause sudden occlusion the bowel. Death may be due to an acute attack. The stone may fo a diverticle for itself in the bowel.

The symptoms of intestinal stone are, as a rule, distinctly chro In some instances there is a history of long-continued digestive d turbances, with occasional attacks of pain and sickness, and w generally some amount of constipation. The patients, indeed, pres the symptoms of a persistent, incomplete, and inert obstruction in t intestine, and are apt to become emaciated and hypochondria Symptoms such as these may continue for years.

B. The chronic cases. — The causes of chronic obstruction of t bowels are very numerous and very varied. For diagnostic purposes t cases may be divided into the four following classes :—(*a*) Stenoses of t small intestine ; (*b*) Stenoses of the large intestine ; (*c*) Fæcal accumu tion ; (*d*) Chronic intussusception.

(*a*) *Stenoses of the small intestine.*—In the matter of *sex* there is nothi clinically to notice. Non-cancerous strictures usually occur about ea middle life ; cancerous strictures are rare before forty. I have, howev met with a case in a woman of twenty-two. Certain strictures of t bowel are congenital.

Previous history.—In non-cancerous cases there will often be a hist of ulcer of the intestine, or of conditions that may lead to destruct changes in the gut ; for example, tuberculosis, dysentery, inju strangulated hernia, perityphlitis, pelvic peritonitis, and so forth.

Course.—The onset is usually gradual. The course of the maladj slow and peculiarly irregular. Acute attacks, due to temporary block of the canal, of obstruction are apt to appear from time to time, a the case is often fatal in an acute obstructive attack. The conte of the small intestine are fluid, and the canal may be much narrow before actual obstruction takes place.

The most conspicuous feature of stenosis of the lesser bowel is t occurrence of attacks of paroxysmal pain at longer or shorter intervals.

The pain is distinctly intermittent, and is of the nature of co Long intervals of absolute freedom from suffering often intervene betw the attacks. As the intervals between the paroxysms shorten, t attacks become of longer duration ; but, in the earlier periods of the ca these intervals may amount to many days or even weeks. The earl attacks of pain are slight, and are usually ascribed to indigestion, flatulen and the like. They are very often provoked by food, and especially by digestible food. The pain radiates from the navel and is seldom localis

It most usually appears some three or four hours after food, rarely later, but not infrequently earlier. As time advances the attacks become more and more frequent, and more and more severe. When complete obstruction sets in, as in a definite attack of acute obstruction, the pain becomes continuous, but with exacerbations.

In uncomplicated cases there is no definite local tenderness.

During the earlier attacks of pain there may be nausea. In the more severe attacks the patients vomit. The vomiting is often provoked by food, it is late to appear, is often scanty, and, except towards the end of an acute obstructive attack, is rarely fæculent. In the interval between attacks of pain the patient either feels well, or merely complains of indigestion, or of some nausea, and trouble with the bowels.

On the whole, it may be said that vomiting is neither pronounced nor very distressing. It is a little more marked the higher up the obstruction. It may be said to exhibit considerable fluctuation.

The state of the bowels is subject to the greatest variation. In about 60 per cent of cases constipation is the prominent feature. In something less than 40 per cent there is constipation alternating with diarrhœa; but in a very few cases only is diarrhœa the ordinary condition of the bowels. During the initial attacks, and during the final attack, constipation is almost invariable, and may remain absolute for many days, or even for two or for three weeks. The constipation at first yields to treatment, but soon becomes more and more obstinate. It is important to note that the earlier attacks are often relieved at once by an aperient. Like relief may follow the use of an enema.

Sometimes an attack of long-continued constipation is suddenly relieved by a copious and spontaneous stool. In such cases, probably, the plug or other obstructing agent has yielded abruptly.

It is not very uncommon for the patient, after days or weeks of absolute obstruction, to pass a copious motion just before death.

In cases of cancerous stricture dark-coloured clots (containing altered blood) are passed in rare instances; less rarely the motions are discoloured by blood.

Tenesmus is practically unknown.

As regards the general condition of the patients, they become progressively weaker as the disease advances, being worn out by the frequent attacks of pain and vomiting, and enfeebled by the loss of appetite that is often a conspicuous symptom. Emaciation is usually pronounced, and the patient's wasted and cachectic aspect may be such as to suggest the presence of malignant disease in cases of a non-malignant nature. In examples of cancerous stricture, emaciation, loss of appetite, and loss of colour are, of course, all much more marked.

The state of the abdomen.—The abdominal walls remain flaccid except during some of the more painful paroxysms, or after the occurrence of peritonitis.

During the attacks of obstruction there will be some meteorism, which, however, is never excessive. In the intervals between the attacks

the abdomen will not be swollen; its walls, indeed, are commonly retracted, especially in cases associated with much wasting and diarrhœa.

It is very usual for the movements of the intestinal coils to be visible through the parietes, a circumstance that is best seen during the paroxysms of pain. This symptom is one of the utmost importance; it indicates the presence of a long-abiding incomplete obstruction and of hypertrophied coils above it.

In no instance among the recorded non-malignant cases was any tumour to be felt, nor any localised dulness that could assist in the diagnosis of the ailment.

In the cases of cancer a tumour has been detected in some 30 per cent of the recorded cases.

Areas of dulness due to an accumulation of fluid in the gut above the stricture are fairly common; equally common is it to obtain, on palpation, splashing sounds as of water in a membranous bag.

Rumbling and gurgling sounds are commonly heard, and are audible to others beside the patient. They are especially noticed during the attacks of pain. These sounds, together with the movements of visible coils, are much increased by purgatives.

Except in the early stages purgatives are often attended by much pain and much commotion within the abdomen. After the bowels have acted, however, there is much relief.

It has been said already that cases of stricture of the lesser bowel are usually attended by certain painful attacks, at intervals, which indicate a temporary obstruction of the strictured part. It may be readily understood that the first occurrence of this obstruction may also be the last—that, in fact, the stenosis, after existing for some time without causing symptoms, may become suddenly occluded, and that this occlusion may bring about a fatal issue. Such a case is reported by Réfrégé. It concerns a man, aged forty-nine, who had been liable for some months to constipation. For some days before his admission into hospital he had had some pain in the lower part of his abdomen. On admission the limbs were cold and cyanosed, the face livid, the eyes sunken, the patient much troubled by vomiting, and the pulse very small and feeble. There was constipation. An epidemic of cholera existed at the time, and the case was taken for an example of that disease. The patient was treated by hot baths and by such measures as were then in vogue for the treatment of cholera. He died on the eighth day after admission. Before his death stercoraceous vomiting had occurred, and the general character of the case had been recognised. The autopsy revealed the stricture in the lower ileum which would barely admit a crow-quill.

With this form of intestinal obstruction—considered purely clinically—must be associated for diagnostic purposes:

(α) Simple and malignant strictures of the small intestine.
(β) Some cases of adhesions binding some portion of the bowel into a fixed loop or fixing it in a bent position.

(γ) Cases of compression or matting together of several coils of intestine by adhesions.
(δ) Obstruction by neoplasms.
(ε) Some cases of obstruction by gall-stones and foreign bodies.
(ζ) Some cases of compression by a tumour outside the gut.

All these forms of intestinal obstruction may present symptoms which resemble one another more or less closely, and which seem to find their typical representation in a case of stricture of the lesser bowel. In each instance it will be noted that there is some permanent but partial occlusion of the intestine. The resemblance between these various forms of intestinal obstruction is very commonly so close that a precise differential diagnosis is impossible. In the second and third forms there will probably be some history of the local peritonitis which gave rise to the adhesions producing the obstruction. In the fifth form there will be the history of gall-stones or foreign bodies. In the sixth variety, the tumour, which will probably have origin in the pelvis, may, in many instances, be obvious upon examination.

(*b*) *Stenoses of the large intestine.*—In these cases the symptoms are very similar to those of stenosis of the small intestine. In the matter of sex and age and previous history there are the same circumstances to be noted which have been alluded to in the previous paragraph.

The course of the malady is attended by a similar irregularity, and by the same kind of paroxysmal attack.

The pain is of the same paroxysmal, colicky character, but, on the whole, is much less severe.

The interval of time between the earlier attacks is often considerable. Thus in one case nine months elapsed between the first and the second. In other instances there have been three or four attacks a year for some years. As the stricture narrows these occurrences become more frequent and more troublesome.

Unlike the strictures of the small intestine, stenosis of the colon is generally unattended by symptoms of the nature of indigestion. There is usually but little connection between the attacks of pain and the indigestion of food.

The attacks in cases of stenosis of the lesser bowel are commonly relieved by the administration of a purge; but in cases involving the colon the opposite usually obtains. Aperients are apt to aggravate existing symptoms—a circumstance that depends, no doubt, upon the more solid character of the contents of the larger bowel.

The final obstruction is usually preceded by many attacks of paroxysmal pain. Between these attacks the patient may feel fairly well, although he is usually troubled by constipation, or by constipation alternating with diarrhœa and with much flatulence. When the obstruction becomes absolute the character of the pain changes, just as in the case of the small intestine; it ceases to be distinctly intermittent and becomes more continuous. Pain in the back is not uncommon in cancer

of the colon, especially in cancer of the sigmoid flexure. In the la stages of the disease it may be severe.

The prevailing condition of the bowels is one of chronic constipatio now and then associated with spurious diarrhœa; just as is seen cases of stricture of the rectum. Of thirteen cases of simple strictu constipation was the prevailing condition in eleven; in the remaini two examples constipation alternated with marked diarrhœa. In t cancerous cases diarrhœa, or diarrhœa alternating with constipation, much more common. In twenty-eight cases of cancerous stricture co stipation was prominent in fourteen; in the remaining fourteen there w diarrhœa.

The final attack is characterised by absolute constipation. Nothi may pass the rectum for ten or twenty days before death. In some ca the period of absolute constipation has exceeded these limits, and h attained a duration of thirty and even of forty days. During the earl attacks there is also constipation.

The constipation at first yields to aperients or enemas, but in ti becomes more and more obstinate. Enemas usually act more efficient than purgative medicines. It has been shown that in some ca water can be injected through the strictures from below, but not fr above.

Vomiting is less marked in stenosis of the colon than in that of t small intestine. In the earlier attacks it may be entirely absent, appear late and be very scanty. In more serious attacks vomiting more frequent, and in the final attack it is constant. It is seldom a d tressing symptom and often fluctuates in severity, being sometimes abs for days, even during the final obstructive attack. It is rarely fæcule except during the obstruction that immediately precedes death. Ev under such circumstances the cases of stercoraceous vomiting are to th of non-fæculent vomiting as five to seven.

Among the more special symptoms tenesmus may be noticed. T is especially apt to occur in cases of stricture low down in the col and particularly in cases associated with diarrhœa. It is more mark in the early than in the later stages of the disease, and is more comm in the malignant than in the non-malignant cases.

In some cases of stricture in the lower part of the sigmoid flex the motions passed have been distinctly flattened or otherwise altered shape. When the stricture is in the higher part of the colon the fæ matter passed through the stenosed part becomes remodelled in the low portion of the bowel as it passes towards the rectum. The shape a size of the motions, therefore, are of very little value in the diagnosis stricture of the colon. The rod-like and tape-like motions nearly alwa depend upon a morbid contraction of the sphincter. The motions a often flattened by a very enlarged prostate. I have known a lar rounded mass of fæcal matter, the size of a hen's egg, to be passed by patient who had a stricture that would only just admit the forefinger, a which lay only six inches from the anus.

Hæmorrhoids are often met with in association with malignant disease low down in the colon.

A bloody discharge from the anus is met with in about 15 per cent of all cases of cancer of the colon (the rectum being excluded); in most of these instances the trouble is low down in the sigmoid loop. When a cancer is situated low down the motions are now and then streaked with blood and mucus.

Ballooning of the rectum may be said to be common in connection with stricture of the colon, especially when the obstruction is low down. The symptom is due to some disturbance in the innervation of the rectum; it disappears under an anæsthetic. It is met with in connection with other intestinal troubles, such as perityphlitis, and when present cannot be said to do more than raise a suspicion of stenosis of the colon; it is not sufficiently definite to be of diagnostic value.

The general condition of the patient in these cases may be expressed in the same words that have been applied to cases of stricture of the lesser intestine.

In malignant disease the loss of weight, strength, and colour is very marked.

The abdominal walls remain flaccid unless some peritonitis have arisen. So long as the bowels act there is but little meteorism, and in cases associated with diarrhœa the parietes may be retracted. As the obstruction becomes more complete the abdomen becomes more and more distended, and in fatal cases there may be a considerable enlargement of the belly by the time that death occurs. The outline of the colon distended with fæcal matter is often very evident, and in any case the distension is most marked in those parts of the abdomen which are occupied by the large intestine. The outline of the colon, moreover, may be indicated by some dulness on percussion, while the region of the small intestine remains tympanitic.

Often large fæcal masses can be felt in the bowel above the obstruction, masses so prominent as sometimes to form very distinct tumours, the nature of which has not always been accurately recognised.

Coils of intestine in movement are very apparent through the parietes, and sometimes these hypertrophied coils are enormous. Movements in the coils are associated with colicky pains, with loud rumbling and gurgling sounds as of the bubbling of gas through water. A coil on percussion may at one moment be resonant, but when a peristaltic wave passes along it, and the gas which caused the resonance is driven out, the fæcal matter lodged in the coil alone remains, and the percussion note becomes dull. Coils distended with fluid, giving the sense of membranous bags filled with water, are often to be made out, especially after the administration of aperients.

In cases of simple stricture no tumour is to be felt; but in cases of malignant disease a tumour is to be detected through the abdominal walls in no less than 40 per cent of the examples.

With this form the following different varieties may be associated for

diagnostic purposes:—Simple and malignant stricture of the colon; compression of the gut by adhesions; obstruction by neoplasms; compression by a tumour outside the gut; some cases of enterolith in the colon.

(*c*) *Fæcal accumulation.*—Obstruction of the bowels by fæcal accumulation is more common in women than in men, is most frequently met with in those who have passed middle life, and is common in the subjects of hysteria and hypochondriasis. A history of dyspepsia, imperfect teeth, irregular and hurried meals, and a "neurotic condition" are common conditions. Such patients are liable to habitual and troublesome constipation; their bowels are seldom opened without the aid of aperient or enema. Many days elapse without a sufficient stool, and from time to time enormous quantities of fæcal matter are evacuated by artificial aid. Sometimes there is a brief interlude of spurious diarrhœa, which depends upon catarrh excited in the bowel above the fæcal accumulation; the catarrh causes a free exudation to be poured into the intestinal canal, this dissolves a certain amount of fæcal matter, which, finding its way beyond the main mass, appears at the anus as a slight watery motion.

In more marked cases the abdomen becomes distended, evacuations are less frequent and more difficult to obtain; the patient complains of a sense of weight and fulness in the abdomen; his appetite is poor, his tongue foul, his breath offensive. He is much troubled by indigestion, by distension after food, by flatulency and by eructations. Not infrequently he becomes much weakened and loses flesh. He may become lethargic and morose, or fretful and uneasy, and may present some phase of hypochondriasis.

If the abdomen become greatly distended other symptoms may appear, such as palpitation, a sense of oppression in the chest, and a little dyspnœa from a pressing up of the diaphragm by the distended bowels. Pressure may be exercised upon the lumbar or sacral nerves, and the patient may complain of discomfort in the genitals, or in the thigh (genito-crural nerve), or down the leg along some part or parts of the great sciatic nerve.

The constipation may remain absolute for weeks or even for months. When this comes about the patient has most probably lost his appetite, he is troubled with frequent and foul eructations, he is greatly distressed by the distension of the abdomen, he suffers from nausea and ultimately from vomiting; the vomiting may become fæculent, although this occurrence is very rare. Yet even when the symptoms have advanced to so extreme a degree, relief may be afforded either by enema or by a spontaneous evacuation, and after the bowel has been emptied recovery may follow. On the other hand the illness may progress from bad to worse; the patient begins to suffer pain in the abdomen, or an increase in the comparatively slight pain that may have existed for some time, and he dies of the effects of the unyielding obstruction.

In several cases, after complete constipation for two or three months, the patient has had a relief of the bowels at the end of that time and has rapidly recovered. Mr. Pollock reports the case of a

lady, aged thirty-five, who had one evacuation of the bowels only every three months, that is to say, four evacuations in the year. Dr. John Blake reports the case of a man, aged forty-six, whose bowels were stopped absolutely for eighteen weeks. At the end of that time he passed a motion spontaneously, but he died in a few days. It is not the least interesting fact in this case that an aspirator-trochar was introduced into the abdomen of the unfortunate person no less than 150 times during the continuance of the constipation, and before the conclusion of the case the patient was taking twelve grains of morphia a day. In another case, reported not long ago, a man, aged twenty-six, who had been always liable to constipation, had at one time no evacuation of any kind from the bowels for the almost incredible period of eight months and sixteen days. Dr. Thomas Strong, who reports this case with considerable detail, refers to instances of patients who suffered from absolute constipation for periods respectively of seventy-six days, fifteen weeks, seven months, eight months, and nine months.

In another and common class of case the patient is liable from time to time to obstructive attacks. In these attacks it is probable that the much-narrowed canal becomes more or less suddenly blocked; whereas in cases of the previous class the occlusion is brought about by very gradual processes. The more abrupt stoppage may be due to the dislodgment of a hard mass of fæces; or it may depend upon bending or kinking of the distended bowel. The latter condition may be met with in the transverse colon and in the sigmoid flexure; especially at the point of junction of the flexure with the rectum.

The most important feature in these cases of fæcal accumulation is the tumour formed by the mass of retained fæces. This tumour is, as a rule, most readily felt in the cæcum. Such tumours feel hard and uneven, are of a globular shape, and are usually painless. Sometimes, however, the region of the tumour is the seat of much tenderness, a circumstance that probably depends upon some ulceration within the bowel, or upon a little local peritonitis. In the ascending colon the tumour will probably feel softer, will be cylindrical in outline, and very like a chronic intussusception, especially as its limits cannot ordinarily be well defined.

Masses in the transverse colon, when near the hepatic flexure, may give rise to the impression that the liver is enlarged, the extent of dulness over that viscus being increased. These tumours, when in a mobile part of the colon, are of course themselves movable. Masses in the transverse colon may cause the gut to become bent down, and in such cases the fæcal tumour has been felt near to the symphysis. When in the descending colon, or sigmoid flexure, the fæcal mass will usually feel harder and its division into scybala may be detected. Indeed, on account of their uneven and nodular surface tumours in this situation have been compared to a large rosary.

In thin persons and in others also when under an anæsthetic, the softer of these fæcal masses may be affected by pressure, and may behave

under the fingers like a mass of dough or of putty; when such character is presented by the tumour the diagnosis of its nature is plac beyond doubt. Fæcal tumours may exist unchanged for weeks or mont and may coincide with the passage of normal motions or with the spuri diarrhœa to which attention has already been directed.

These tumours have been mistaken for cancer, for sarcoma of t peritoneum, for chronic intussusception, for tumours of the liver, g bladder, stomach, spleen, and kidneys, for ovarian and other pel tumours, and for pregnancy. The great distension of the abdomen a the presence of much flatus within the intestines in these cases are apt obscure the details of the mass when it exists.

(*d*) *Chronic intussusception.*—The anatomical form of intussuscepti most often met with in chronic cases is the ileo-cæcal; it for more than one-half of all the examples. The enteric form is the vari that is least often chronic. The relative proportion is thus given Rafinesque, his conclusions being based upon a collection of fifty-f distinctly chronic cases:—

Ileo-cæcal	61 per cent.
Colic	15 ,,
Enteric	14 ,,
Ileo-colic	10 ,,
	100

The clinical features of chronic intussusception are usually v ambiguous. No form of intestinal obstruction presents so many confus elements in the diagnosis; no form has led to more conspicuous errors the right appreciation of the nature of the malady.

Out of the fifty-five cases collected by Rafinesque many were ne suspected to be examples of intussusception, and no less than twen seven were the subjects of an absolutely incorrect diagnosis. Chro intussusception has been mistaken for fæcal accumulation, for rec polypi, for cancer of the bowel, for ulcer of the stomach, for dyspep for chronic dysentery, for gastro-enteritis, for tuberculous peritonitis, a for other ailments equally remote from the actual nature of the disease

The course of the malady may extend over many months, and m be protracted even for a year.

During its progress the malady usually follows a most irregu course. At one time constipated, the bowels may be at another ir state of diarrhœa. There may be violent pain one day and none next. Some patients are troubled by severe vomiting, others never sick. In some cases there are long intervals of freedom fr sickness, in others there are no such breaks.

The onset of the malady is usually a little indefinite, and the earli symptoms are often ascribed to indigestion, to colic, or to simple irre larities in the bowels. In about 30 per cent the onset has been abru the case assuming a chronic aspect subsequently. In any case pain usually the first symptom.

The pain that occurs during the progress of the disease is paroxysmal Attacks of pain may appear several times a day, or but once in the twenty-four hours. Sometimes days and even weeks have elapsed between the paroxysms. The intervals between the attacks are seldom regular, and when the pain does appear at stated times the occurrence is probably due each time to a repetition of the same cause. Attacks of pain become shorter and the pain itself more diffused. In the less protracted cases the suffering may be almost continuous, marked, however, by exacerbations.

The pain, when present, has the general character described when dealing with the acute form of the disease.

Vomiting is not a very conspicuous symptom. In forty of Rafinesque's cases where this symptom is mentioned it occurred, more or less frequently, in twenty-four instances. In four instances the patient was sick at rare intervals, in seven vomiting did not appear until a few days or hours before death, and in three cases there was an entire absence of vomiting throughout the progress of the malady. The attacks of sickness were very rarely continuous; they usually appeared at irregular intervals, coinciding with the attacks of pain or depending upon some alimentary excess.

The appetite usually becomes much impaired, and the symptoms are often aggravated by food. In a large number of instances it showed considerable fluctuations, and in one case at least it was voracious.

The state of the bowels is most variable. Natural and regular stools may be passed during the greater part of the disease, or there may be long-continued diarrhœa, or marked constipation, or alternations between the two last-named conditions. Indeed, the only certain feature in the state of the bowels in chronic invagination is the feature of uncertainty. On the whole, a tendency to diarrhœa is the most common condition, and a normal state of the bowels the rarest.

Blood is passed with the stools in about 50 per cent of the cases, and tenesmus is present in 13 per cent.

In chronic invagination the bowels usually respond to the action of aperients; these drugs sometimes give relief, but more often they provoke at least a temporary aggravation of the symptoms.

In any case of long-standing intussusception a certain degree of persisting obstruction must exist in the intestine. As a result of this, the bowel above the invagination becomes hypertrophied. The patients, on the other hand, usually emaciate, and the anterior abdominal parietes share in the general wasting. Thus it happens that coils of intestine are very often to be seen in movement beneath the belly wall, a circumstance that will be most distinct when peristaltic waves are passing along the disordered intestine.

The general condition of the patients in chronic invagination shows, as may be supposed, considerable variation. In the early periods of the disease, and in the intervals between attacks of pain, they may appear to be in fair health. In time, however, they usually become anæmic and

emaciated; they are worn out by the frequent pain, and exhausted b the vomiting and diarrhœa. Some die of an acute attack that suddenl appears and puts an end to the case; others die simply of exhaustio and marasmus; a few succumb to perforative peritonitis, and a sma number to effects depending upon the spontaneous elimination of th intussusceptum.

With regard to the state of the abdomen, little can be added t what has been said concerning the acute form of the malady. As a ru the abdominal walls remain flaccid and present no abnormal featur When a long-continued constipation exists there may be some meteoris which will, however, always be moderate in degree. Tenderness o pressure is very seldom observed unless peritonitis be present.

A tumour is to be found in about one-half of the cases. Its characte have been fully described (page 850). Among the fifty-five cases collect by Rafinesque the tumour was felt in the rectum in seven instances, a had protruded beyond the sphincter in nine.

C. The cases where acute obstruction supervenes upon sympto of chronic obstruction.—There is no one of the many forms of chro obstruction described in the preceding paragraphs in which all t evidences of acute occlusion may not arise.

If the acute obstructive attack come on while the case is und observation there can be no difficulty in the diagnosis; if, however, t patient is seen for the first time during the height of one of su attacks, then the symptoms may be very readily attributed to one other of the pathological conditions that lead to acute strangulatio Thus the abdomen has been opened under the impression that a coil intestine was strangulated by a band, when the primary cause of t occlusion was found to be a malignant stricture of the bowel. There no one special form of intestinal obstruction that can be placed in th class and in no other. There is no form of chronic obstruction of t bowels that invariably leads to an acute attack.

The most common varieties of chronic obstruction are those that depe upon fæcal accumulation and upon stricture of the colon. In the form case a kind of intestinal obstruction that often presents very pronounc and violent features may appear at any moment. In connection wi strictures, also, the changes may proceed quietly for months, the stenos part becoming narrower and narrower, and the symptoms more and mo clearly defined; suddenly the patient is seized by an acute attack obstruction; and, if death result, the gut will be found to have beco suddenly occluded at the narrowest part by kinking, or the strictu blocked by some foreign substance, by a fæcal mass, or by a mass undigested food. If the stricture involve the upper parts of the rectu the distended sigmoid flexure above the stenosis may have become twist upon itself, and thus have brought about the condition of volvulus.

Again, in the less common forms of chronic obstruction the same co ditions may be met with. Thus, chronic intussusception very often en

in an acute attack which may prove rapidly fatal. Coils of intestine matted together by adhesions may become suddenly occluded by bending or kinking, at one or more points, and so lead to acute manifestations. A case of chronic volvulus, or of volvulus associated with slight symptoms, may, as a result of distension or of paralysis, become at any moment an example of acute volvulus with the appropriate symptoms. Any portion of the bowel partially occluded by compressing adhesions, or by a tumour outside its walls, or by a neoplasm or a foreign substance within its canal, may become completely obstructed in a moment by any of the causes of the sudden occlusion of strictures.

The patient may have many of such attacks, and these very often exhibit an increasing degree of severity.

With regard to the diagnosis between these quasi-acute attacks and cases of acute strangulation of the bowel, such as may be due, for example, to adhesions, the most important factor is the patient's past history. There will be usually a history of the symptoms incident to chronic obstructions, and of previous attacks of like character but of less pronounced severity. These attacks are distinctly less abrupt and less violent than in acute strangulation. The pain is usually by no means so severe, nor is the condition of prostration so marked. To one sign, however, in the differential diagnosis too much importance can scarcely be attached. It is this. In the acute attack supervening in a chronic case, the coils of intestine may be visible through the thinned parietes, and this symptom will be absent in cases of primary acute obstruction. In the former variety the symptom may be lost sight of if the meteorism become extreme, or if peritonitis occur; and it may be rendered much less distinct if the peristaltic movements have been moderated by the use of opium.

There are, unfortunately, a few rare cases in which the presence of a partial obstruction of the intestine is revealed for the first time by an acute attack. That is to say, a stricture has existed in the intestine (probably in the small intestine), but has not yet so narrowed the lumen of the tube as to cause symptoms. On a sudden, however, the stenosed part becomes blocked by a mass of undigested food, or the bowel becomes occluded by kinking at the seat of stricture, and symptoms are thereby produced that assume an acute character at once. An acute attack occurring under these circumstances may be fatal; and cases are recorded where a stricture of the small intestine revealed itself by one attack of rapidly advancing obstruction ending in death.

General diagnosis of intestinal obstruction.—Almost any acute trouble within the abdomen during the first few hours of its existence may be mistaken for intestinal obstruction; but most of the cases clear up in the course of time. [See the remarks on "Peritonism" in the article on "Peritonitis," p. 616.] In every suspected case of intestinal obstruction the previous history should be fully investigated. The abdomen should, of course, be carefully examined, and as a matter of routine an examination should be made of the hernial orifices and of the rectum.

The following are the common diseases which have been mistaken for intestinal obstruction :—

Peritonitis, especially the localised form, due to mischief in the appendix. The main points in the distinction of these diseases are the following :—

In peritonitis the temperature is usually raised when the patient is not in a condition of collapse; in acute obstruction it is nearly always subnormal. In peritonitis the pain is more continuous, and is less like that of colic; it is increased, rather than relieved, by pressure. In acute obstruction the initial pain is commonly the most severe; in peritonitis the pain steadily increases up to a certain point. In peritonitis the vomiting is less urgent, less copious, less frequent, and practically never really fæculent; the constipation is less absolute. In peritonitis the skin of the abdomen is hyperæsthetic, the belly wall is hard and board-like, and there is great tenderness on pressure; the belly, indeed, is not distended, but smooth and firm. No peristaltic movements are evident in peritonitis, the patient is usually less restless, and more inclined to keep the knees flexed and the hands above the head (see p. 613).

Cholera.—This disease has been imitated by the most acute forms of intestinal obstruction; in these cases the patient may rapidly fall into a state of cholera-like collapse; the extremities become cool, the surface cyanosed, the pulse thready and almost imperceptible, the voice sunk to a whisper, and the countenance Hippocratic. At the same time there are violent vomiting, cramps in the limbs, suppression of urine, and extreme prostration. The cases that have most closely resembled cholera have been cases of very acute strangulation of a considerable portion of the small intestine, especially of the upper parts of that bowel. The strangulation may have been preceded by profuse diarrhœa, or the gut below the obstruction may have been emptied by diarrhœa after the strangulation had occurred. In many instances of wrong diagnosis the cases had occurred during an epidemic of cholera.

Another form of obstruction that may resemble cholera is ultra-acute intussusception, which may be associated with much purging.

Dr. Barlow mentions an instance of the patient being thrown into a choleraic condition by obstruction due to masses of undigested food.

An excellent discussion of the chief features in the diagnosis of these cases has been afforded by M. Félix Réfrégé. He deals with fourteen cases of error in diagnosis. In only four of the fourteen cases were cramps in the limbs noticed, and in all, save two, there was absolute constipation.

There can be little real difficulty in the diagnosis if too hurried an opinion be not given. The obstruction attacks are associated with intense pain at the beginning, attended by constipation; in cholera, pain and profuse diarrhœa are absent. The abdomen soon becomes retracted in cholera, but meteoristic in acute strangulation. Vomiting does not set in quite so early in cholera as in cases of acute obstruction; in many

cases it is entirely absent, and when present is non-fæculent, and has the peculiar whey-like appearance so often described.

Lead colic.—A case is reported by Dr. Fagge of a man, aged twenty-nine, who had a blue line on the gums, but whose intestinal symptoms were due not to lead poisoning, as was at first supposed, but to partial obstruction from shrinking of the mesentery. On the other hand, I have known lead colic mistaken and treated for intestinal obstruction.

Poisoning by arsenic.—In several instances cases of acute strangulation have excited suspicions of poisoning by arsenic, and the doubt has only been cleared up at the autopsy. Leichtenstern alludes to several examples.

Hepatic or renal colic may be imitated by acute obstruction in the upper portion of the small intestine associated with profuse non-fæculent vomiting, intense paroxysmal pain, collapse, and a retracted condition of the abdomen.

Meningitis.—Dr. Fagge refers to a case of acute obstruction of the jejunum where, on account of the delirium, the vomiting, and the retracted abdomen, meningitis was suspected.

Sarcoma of the omentum has simulated true obstruction, and the tumour formed has been at first mistaken for a fæcal mass.

A *cyst of the mesentery* has been mistaken for an intussusception tumour, and an intussusception tumour for a new growth or a mass of fæcal matter.

Tumours formed by fæcal masses have been mistaken for many other affections (see p. 868), and notice has already been taken of the many diseases which have been confused with chronic intussusception (see p. 868).

The confusion between acute or subacute intussusception and *dysentery* or *enteritis* has been of frequent occurrence.

Prognosis and terminations in intestinal obstruction.—In genuine cases of acute intestinal obstruction the prospect of spontaneous relief is so extremely slight that this possibility—which has been exaggerated by imperfect reports of cases and errors in diagnosis — may be disregarded when the question of treatment is considered. Except in quite rare instances, all the cases, if untreated, end in death. The prospect of spontaneous recovery in acute intussusception has already been referred to.

The average duration of a case of strangulation by bands and the like (from onset to death) is five days, of volvulus of the sigmoid flexure six days, and of acute obstruction by gall-stones seven days. The most acute cases of intussusception may end fatally in twenty-four hours. The ordinary acute cases of intussusception may be expected to end in death in two to seven days, and the subacute cases in seven to thirty days.

Cases of chronic obstruction may be extended over many months. Patients with stricture of the bowel, if carefully treated by medical measures, may survive for eighteen or twenty-four months. The course of a case of fæcal accumulation is still more indefinite. In general terms it

may be said that the probable duration of life in a case of chron obstruction, when once the obstruction symptoms have become definite, i from three to six months.

Death is most usually due to peritonitis or to that form of septi intoxication which marks the less vigorous and sthenic phases of the inflammation.

Ulceration and perforation of the bowel are common. The strangu lated loop or the intussuscepted gut may become gangrenous. The over distended bowel may give way. Enteritis appears occasionally, whil in a small proportion of cases death is due to a septic pneumonia. I the most chronic cases the patient may die of marasmus and exhaustio worn out by the continued pain and vomiting, and starved to death fro the inability to assimilate food.

The account of the treatment of intestinal obstruction which follow is derived mainly from the author's article in the *System of Surgery*.

Treatment of acute intestinal obstruction.—(*a*) *Strangulation bands, etc.*—The treatment of internal hernia is dealt with on p. 808.

It is needless to say that in this and other forms of acute obstructio the patient must be kept absolutely at rest. Collapse may be met l warm bottles, and the intense pain by hypodermic injections of morph but it must be remembered that morphia will tend to mask the symptom under its use the pain lessens, the pulse improves, the expressi becomes less ghastly, and the vomiting may, for a while, be put abeyance. No food should be given by the mouth. It is useless. Thi may be relieved by sucking a little ice and by rectal injections of war water. In cases in which the thirst has been maddening I have seen objection to allowing the patient to take now and then a copious draug of fluid (hot if possible). He almost immediately rejects it, but it serv in a way to wash out the stomach, and makes the man for a little wh more comfortable and more contented. Flannels wrung out in h water and applied to the abdomen may do something to relieve t pain. The mouth should be washed out occasionally with an antisep solution, and be kept clean; much distress is often caused by the foulne of the mouth.

There is but one measure of active treatment, and that is abdominal section. The sooner the operation is carried out the bett and it should be done as soon as the diagnosis is established, and as so as the first symptoms of shock have passed off a little. The operation opening the abdomen is in itself a small matter; delay is a fatal matt To temporise is quite useless. In this form of obstruction spontane relief, while not absolutely impossible, is yet excessively rare, much rar for instance, than the spontaneous relief of strangulated hernia. T operation consists in opening the abdomen, in seeking the cause of t obstruction and in relieving it. The difficulties likely to be present in this form of obstruction are not many. Bands are ligatured a divided; diverticula are probably removed; apertures and slits are open up, and the bowel freed.

It is not, however, the actual strangulation of a loop of gut which is leading directly to the patient's death at the time of the operation. If he dies, he dies from septic poisoning, and the septic matter is derived from his own intestines. It is desirable, therefore, in these acute cases that an opening should be made in the distended bowel and the contents allowed to escape. The mortality of the operation for acute intestinal obstruction is very high, possibly 75 per cent; but it is becoming lower, and will no doubt be materially reduced when operations are performed earlier, and are not considered complete until the bowel has been emptied.

In the most urgent and advanced cases of all—the neglected cases—it is desirable to do as little as possible; and the best results (such as they are) have attended the opening of the abdomen without an anæsthetic, and the rapid establishment of an artificial anus in the first coil of distended bowel met with. This crude measure has been attended with no little success. When the condition of the patient will allow, the washing out of the stomach with hot water after the operation is of very material service. At any time it gives marked relief, but, although enthusiastically advocated by many surgeons, it is, of course, a palliative measure only.

(*b*) *Volvulus of the sigmoid flexure.*—There is no evidence to show that spontaneous cure ever follows this affection when the twist is complete and well defined. Unless relieved the condition would appear to be always fatal. General measures of treatment have been alluded to in dealing with obstruction by bands. The abdomen should be opened and the coil emptied.

It is seldom possible to unfold the volvulus while the gut is distended, although an attempt may be made in that direction, and such attempts have been successful. It is better to establish an artificial anus in the summit of the loop. The mortality of the operation has been high; but it gives the patient his only chance.

(*c*) *Acute intussusception.* — In addition to the general measures of treatment, opium, or morphia, is very necessary in these cases to stop all peristaltic movement. An attempt may be made (especially in children) to reduce the invagination by means of air introduced into the rectum. This is most conveniently done by means of Lund's insufflator, and is carried out under ether. In the place of air water may be used, and in this case the child's pelvis should be much raised. If the invagination be reduced by these means it is very apt to re-form. Inversion of the body is a violent measure, which neither on theoretical nor on practical grounds has anything to recommend it. Failing reduction by the insufflator, the abdomen should be opened at once. There is no excuse for delay; the longer the case is left the more difficult does reduction become. In more than one-half the reported cases of operation reduction was found impossible. If the intussusception be reduced it is well, the abdomen may be closed. Failing reduction four courses are open, or rather have been advised:—

(i.) To cut away the whole of the invagination, and to unite the two ends of the divided bowel by suture. This is only suited for small invaginations, and for cases in which the patient's condition is still good.

(ii.) To cut away the invagination and establish an artificial anus. This measure has been attended with some slight success.

(iii.) To leave the intussusception untouched, and to establish an artificial anus above it. This procedure is not satisfactory. It leaves the invaginated bowel to progress, probably toward gangrene, and encourages conditions which will render the fæcal fistula permanent.

(iv.) To establish a communication between a loop of bowel above the obstruction and a loop below. This measure, known as short-circuiting or intestinal anastomosis, leaves the invagination alone, and has at present few arguments to recommend it.

It must be confessed that none of the four procedures just mentioned has been attended with any encouraging success; they must be ranked as quite desperate measures, forced upon the surgeon by delay in operation.

Braun has collected 63 cases of operation for invagination. In 51 attempts at reduction were made, and in 26 the gut was reduced. Out of the 51, 40 died and 11 recovered. In 10 cases resection of the bowel was performed, but no patient recovered.

Dr. Wiggin has collected 103 records of intussusception treated by various measures. Of 39 treated by enema or insufflation 23 died and 16 recovered. Of 64 treated by laparotomy 43 died and 21 recovered. These figures are of little value in affording guidance as to the danger of the operation, inasmuch as nearly all the successful cases are recorded while the fatal cases are left to oblivion.

There is a slight prospect of recovery in intussusception by spontaneous means, namely, by gangrene of the intussusception; and when in an acute case days have been allowed to elapse, the prospect of success by operation is so slight that, so far as statistics at present indicate, it is better to leave the cases "to nature." If the enormous number of unpublished cases of late operation in intussusception could be brought to light, this course would receive definite support.

Elimination of a gangrenous intussusception takes place in about 42 per cent of the cases, and the death-rate among those in whom it does occur is over 40 per cent.

The prospect of spontaneous recovery, therefore, is small, but it would appear so far that the prospect of recovery from a late operation is smaller. An operation to be of service should be performed at once, and without any delay when once insufflation has failed.

(*d*) *Acute obstruction by gall-stones, foreign bodies, etc.*—In certain of these cases relief has attended the free administration of opium, followed after a while by copious and repeated enemas.

In the acuter cases laparotomy should be performed. The measure has been attended with considerable success in cases treated early.

Treatment of chronic intestinal obstruction.—*Stricture and stenosis of*

the intestine.—Much can be done in these cases by strict attention to dieting, and the selection of such easily-digested foods as will leave little débris in the bowel. Pepsine, papain, and other artificial digestives are of value. The bowels must be kept acting by laxatives and frequent enemas. No advantage attends the use of the so-called long tube; this tube cannot be passed beyond the sigmoid flexure, as I have many times demonstrated. Massage is often of considerable service in maintaining an action of the bowels. Salol or other intestinal antiseptic will be found of service in lessening the tendency to decomposition in the bowel. As far as his movements are concerned, the patient must regard himself as an invalid.

If there be much pain opium may be called for, but its use is in every way to be deprecated, as it undoubtedly increases the tendency to obstruction. When the pain becomes sufficiently severe to need the frequent administration of opium the time for an operation is at hand.

Sooner or later operative measures will be called for:—

These are represented by (*a*) the establishment of an artificial anus; (*b*) the resection of the bowel at the strictured part; (*c*) the establishment of an intestinal anastomosis.

(*a*) The establishment of an artificial anus is not adapted for strictures in the small intestine, except as a means of warding off impending death. An artificial anus high up in the small intestine soon leads to death from malnutrition. This operation (enterotomy or enterostomy) has been usually carried out in cases which, after being for some time chronic, had become suddenly acute.

The establishment of an artificial anus in the colon (colotomy or colostomy) is a measure attended with a very fair degree of success. A patient may live for years with an artificial anus in the sigmoid flexure, and in many cases of malignant disease a colotomy is all that can be done.

The risk of the operation depends upon the seat of the obstruction. Iliac colotomy in cancer of the rectum carries with it an immediate mortality of about 3 per cent. Iliac colotomy on the right side performed for some obstruction in the colon—for example, at the hepatic flexure—is attended with a very much higher mortality, probably 20 per cent.

As to the duration of life after colotomy for malignant disease, among 46 patients who recovered from the operation 12 died within six months, and 15 between six and twelve months; 10 lived to some time between the first and second year; 8 died between the second and third year, and 1 died before the fourth year.

In comparing iliac with lumbar colotomy, the advantages claimed for the former are that the operation is more easily and more readily performed, that the bowel can be examined and a diagnosis made, and that the situation of the artificial anus is no more inconvenient than when placed in the loin. Lumbar colotomy is sometimes to be preferred in cases of obstruction attended with very considerable distension of the abdomen.

(*b*) The resection of the bowel at the strictured part, with immediate suture and closing of the abdominal wound, is, of course, an ideal measure; it is rarely possible, however. In one series of cases the disease is too extensive, as in examples of cancer where the disease has spread beyond the gut, or in cases in which the bowel is involved by many and extensive adhesions; in another series the obstruction is advanced, and the patient's condition not such as to allow of a long operation. In very many instances, therefore, and more especially in connection with cancer of the colon, an artificial anus has been established at the seat of the resection. Kendal Franks has collected 51 cases of colectomy for cancer, with a mortality of 40·8 per cent.

The amount of gut removed has usually been a few inches. Koeberlé removed six feet of the small intestine for multiple stricture with success.

(*c*) The establishment of an intestinal anastomosis is an alternative to an artificial anus. A coil above the obstruction is made to communicate with a coil below. The strictured part is thus eliminated from the intestinal canal. This measure is especially indicated in such conditions of disease in the intestine as are not capable of being removed by operation. As an alternative for colotomy it may often be entertained. The operation has been attended with quite encouraging results.

Fæcal Accumulation.—The treatment of chronic constipation need not be considered in this place. The whole matter is dealt with in the section on Constipation (page 696).

In the present chapter we are concerned only with cases when the occlusion has become more or less complete, and pressing symptoms have appeared. In such cases aperients do more harm than good. Abdominal pain may be relieved by opium or belladonna and by warm fomentations. The diet should be restricted to the simplest food, and to that of the most digestible kind. Milk should be avoided. An enema should be administered twice daily. It should be given, when possible, in the knee and left shoulder position, and is readily administered by Lund's insufflator, by which considerable pressure can be exerted. Nothing is to be gained by the use of the long tube. Insufflation of the rectum with carbonic acid has been employed by some surgeons, others advise the swallowing of a large quantity of metallic mercury, a measure which has been followed by some success, but also by some calamities. Massage answers well in many cases, especially in the least urgent forms. Electricity is much advocated by some physicians. As soon as action of the bowels is obtained the movement may be followed by the cautious use of aperients.

It is possible that in certain of the most urgent and most obstinate cases iliac colotomy may be called for; but the cases in which such a measure is indicated must be extremely rare, and in such cases it is probable that some complication exists.

Chronic intussusception.—Great attention should be paid to the dieting of the patient. Benefit of a temporary character attends the use of

enemas and gentle laxatives with the administration, when pain is present, of belladonna with or without opium. Attempts to reduce the invagination by insufflation or injections of water are not likely to succeed. Rectal intussusceptions have been excised per anum with success. In invaginations higher up the only measure is abdominal section. If the intussusception can be reduced—as is very improbable—that is well; failing this the mass should be excised; several successful examples of this operation have been recorded.

FREDERICK TREVES.

PERITYPHLITIS

DEFINITION AND NAMES.—By perityphlitis is understood a peritonitis localised in the region of the cæcum. Many names have been applied to this condition, which serve to perpetuate current views of its pathology. The fact that the effusion in this form of peritonitis is nearly always circumscribed, led early to the belief that it is extraperitoneal, and located in the connective tissue behind the cæcum. In early days, therefore, this affection was known as "iliac phlegmon," and later the more precise term "paratyphlitis" conveyed the same view of the position of the inflammatory changes. When these names were in use it was erroneously assumed that the cæcum had a non-peritoneal surface which brought it in contact with the connective tissue of the iliac fossa, and when the etiology of the trouble came to be investigated it was assumed that the mischief started in the cæcum itself. Upon this belief was based the term "typhlitis"; that is to say, an inflammation of the cæcum itself. This name appears to have been first used by Albers in 1838. In Copland's *Dictionary of Medicine*, published in 1834, although the cæcum and the supposed connective tissue around it are still pre-eminent in the pathology, yet it is recognised that the trouble may start in the appendix vermiformis. Burne seems to have been the first to develop the modern doctrine of the predominant part played by the appendix in this affection. Fitz in 1886 (9) placed the pathology of perityphlitis upon a sound basis, and demonstrated the part of the appendix in its production. Finally, the uncouth name "appendicitis" has been given to this disease.

The name "perityphlitis" does not denote the seat of origin of the malady, but indicates with sufficient clearness the predominant pathological feature of an affection which may arise in more ways than one, and which has no precise clinical individuality until the peritoneum in the cæcal region has become inflamed.

The literary history of perityphlitis is well given by Dr. Kelynack in his work on the *Pathology of the Vermiform Appendix* (Manchester, 1893).

NORMAL ANATOMY.—The cæcum is that part of the large intestine which lies below the level of the ileo-cæcal valve—that is to say, below the point of entrance of the ileum. It is always entirely covered by peritoneum, and is never attached by areolar tissue to the iliac fascia. The mesocæcum, formerly described, has no existence. The cæcum is usually found lying upon the psoas muscle, and is so placed that its apex just projects beyond the inner border of that muscle, and corresponds with a point a little to the inner side of the middle of Poupart's ligament. In exceptional instances the cæcum may be found within the pelvic cavity, or even to the left of the median line. In examples of imperfect descent of the caput coli this part of the bowel may be met with under the umbilicus, or close beneath the liver, or high up in the right loin. There is no reason why perityphlitis should not be met with in any of these unusual situations.

There are certain peritoneal fossæ about the cæcum, but they possess little or no pathological importance; they may seem to limit inflammatory effusions. Now and then the diseased appendix has been found lodged in one of these fossæ, usually in the inferior ileo-cæcal.

The vermiform appendix is very variable. Its average length is about 3½ inches. It may be represented by a mere stump some half-inch in length, or on the other hand may measure no less than 9 inches. It is usually found twisted upon itself, and this condition is due mainly to the shortness or scantiness of its mesentery. The meso-appendix rarely extends more than one-half or two-thirds of the way along the little organ, and is liable also to much variation.

The position of the appendix varies considerably. In the adult body the undisturbed appendix will most often be found behind the end of the ileum and its mesentery, and to point in the direction of the spleen. Another locality not infrequent is a vertical position behind the cæcum or to its inner side. When diseased and exposed by operation the appendix is often to the outer side of the cæcum. The process may occupy the pelvis, and may be found in contact with the sigmoid flexure, the rectum, the uterus, or the bladder. Or it may be found to have entered a right inguinal or a right femoral hernia.

The structure of the appendix is, in general terms, like that of the cæcum. It has, however, the important character of a narrow lumen and a thick wall. The valve of Gerlach, which was supposed to protect the orifice of the appendix, has no existence. The orifice of the appendix—that is, the point of its opening into the cæcum—may be a mere pinhole, or may admit a No. 5 or No. 7 catheter. One very common result of disease is to narrow or obliterate (in whole or in part) the lumen of the little process.

The mucous membrane is liberally supplied with Lieberkühn's glands.

but the most noteworthy feature in the anatomy of the appendix is the very large quantity of lymphoid tissue found in its walls. It may be evenly distributed, or arranged in follicles; and it is always most abundant in children. This collection of adenoid tissue has been compared to that which constitutes the tonsil. Probably it has the same function; that is, to supply leucocytes to repel a bacterial invasion.

The calculi sometimes found in the tonsil may be compared with those found in the appendix.

The so-called muscular coats of the appendix are very thick, the inner or circular layer being the more substantial and the better marked. These coats are made up, however, mainly of fibrous tissue; there is a little muscular tissue found in the inner layer, and a trace in the outer layer, but the amount altogether is very small.

In conclusion it may be pointed out that the appendix represents the terminal part of a long, curved, and conical cæcum, such as may be seen in many apes. It is evident that it is disappearing from the mammalian series. It is a functionless and useless relic, and is the least constant part of the alimentary canal. The diseased conditions which so frequently attack it tend to bring about its obliteration, and it would appear as if its anatomical elimination was being hastened by pathological troubles.

MORBID ANATOMY AND PATHOLOGY.—The peritonitis, which is the essential feature of perityphlitis, may take its origin from morbid conditions within the cæcum or the appendix. It may be said at once that in the vast majority of cases the trouble begins in the appendix, and that perityphlitis due to a primary lesion of the cæcum is undoubtedly rare. The pathology of the affection may be considered under the following headings:—

A. Perityphlitis taking origin from the cæcum.

B. Perityphlitis taking origin from the appendix. (*a*) With catarrh or ulceration of the appendix. (*b*) With gangrene of the appendix. (*c*) With stricture or occlusion of the appendix. (*d*) With actinomycosis of the appendix.

C. The Peritonitis.

D. The Abscess.

A. Perityphlitis taking origin from the cæcum.—The cases that come under this category are, as already stated, very uncommon. Certain writers have gone so far as to assert that perityphlitis of cæcal origin does not exist. Others state that this variety is so rare that not more than one instance of it would be found among some 200 examples of perityphlitis.

Einhorn, as the result of a study of 18,000 post-mortem examinations, states that perityphlitis is of appendicular origin in 91 per cent of the cases, and that in the remaining 9 per cent it is due to primary perforation of the cæcum. Surgical experience would lead us to place the appendicular cases at a higher percentage of frequency.

Osler records two cases of perforation of a cæcal ulcer leading to a perityphlitic abscess. Fitz gives three cases of such perforation—one was due to a fish bone, one to a pin, and a third was ascribed to strangulation of the cæcum.

As an exemplary case of perityphlitis due to trouble in the cæcum, the following may be cited :—

A carpenter, aged nineteen, was admitted into my wards at the London Hospital on 4th July 1894 with perityphlitis. He had been long troubled by constipation. On 1st July, while lifting a heavy weight, he felt a sudden pain in the right iliac fossa, became collapsed, and in thirty minutes vomited. He was sick four times. The bowels had not acted for three days previously. He presented all the symptoms of perityphlitis attended with diarrhœa, and a temperature of 99° to 101° F. An abscess formed in the cæcal region, and was opened on 7th July. The pus had a fæcal odour, and was considerable in amount. The appendix was perfectly normal in every particular. Some lymph was found upon the cæcum at one spot, but it was not disturbed. On 15th July fæces began to escape from the wound, and a fæcal fistula was established. The man ultimately did well.

Perityphlitis of cæcal origin depends, no doubt, upon ulceration of the cæcum. Such ulceration may be caused (*a*) by a sharp foreign body; more than one case of perityphlitis has been found to be due to the impaction of a pin in the cæcum; (*b*) by epithelioma of the cæcum. This condition was illustrated by the case of a medical man under my care who had an ordinary subacute perityphlitis. An abscess formed, and was opened. The appendix to all appearance was quite normal, but there was a perforation of the cæcum. It was shown later that this perforation was at the seat of an epitheliomatous growth. The patient was engaged in active work up to the time of the attack of perityphlitis, although he was wasting, was feeling ill, and had had indefinite trouble with his bowels. The ulceration of the cæcum may further be due to (*c*) tuberculosis, (*d*) typhoid fever, (*e*) dysentery, and (*f*) actinomycosis.

I have operated upon a case of perityphlitis, in a young lad, due to tuberculous ulceration of the cæcum, in which the appendix was to all appearance perfectly normal, and was free from the adhesions which involved the cæcum.

So far as I am aware, in all the cases of perityphlitis ascribed to typhoid fever, the appendix was the seat of the actual perforation and not the cæcum. Ulceration of the cæcum in dysentery does not appear to lead to perityphlitis. Actinomycosis is dealt with on p. 86.

Finally, the commonest cause of such cases of perityphlitis as arise in the cæcum is (*g*) the stercoral ulcer. The stercoral ulcer is due to the mechanical pressure and the chemical irritation of fæcal masses which have long been lodged in the cæcum. Precisely similar ulcers are produced in the appendix by the lodgment of little masses of fæcal matter in that tube. In cases of considerable fæcal impaction the greatest

strain falls upon the cæcum, and in absolute obstruction of the colon low down, the bowel, if it gives way, will give way in the cæcal region.

Long-impacted fæcal masses in any part of the colon give rise to catarrh. This catarrh when of severe degree serves to produce the spurious diarrhœa which is seen in cases of obstruction of the lower colon. This catarrh often passes on to ulceration. It is by means of this catarrh that impacted fæcal masses are often dislodged. Inasmuch as fæcal impaction is most common in the cæcum, it is no matter of wonder that the stercoral ulcer should be most common in that part of the colon.

If the ulcer acquires a sufficient depth to allow the peritoneum to be infected, perityphlitis results. To produce this condition it is not necessary that the cæcum should be actually and freely perforated. The following case serves to illustrate this point. A man of fifty, with advanced carcinoma of the sigmoid flexure, had the whole of the colon above the stricture occupied by retained fæcal matter. In such cases of obstruction the strain, as already said, falls mainly upon the cæcum ; and this patient became the subject of a well-marked attack of acute perityphlitis. An abscess followed, which was incised in due course. The contents were very foul, but no fæces had escaped. Lymph covered the cæcum, but the appendix was apparently quite sound.

It is perhaps needless to say that fæcal accumulation may be excessive, and may even cause acute inflammation of the cæcum, and yet not produce the symptoms of perityphlitis. Thus Dr. Harley reports the case of a youth whose colon was blocked from cæcum to anus with fæcal matter. The mucous membrane of the bowel was inflamed and purulent. The lad died with septic symptoms, but there were no evidences of perityphlitis.

I have met with examples of localised peritonitis about the hepatic flexure of the colon leading to suppuration in patients whose bowel contained masses of scybala, and in whom the acute abscess with its fœtid contents could only be ascribed to a stercoral ulcer.

While claiming a definite position for the stercoral ulcer in the production of perityphlitis it must be owned that the appendix may be the cause of perityphlitis and yet appear to be quite normal to the eye. Many such cases have been reported. I have met with such examples, but have found the apparently healthy appendix to be diseased when subjected to minute examination. I would especially point out, however, that in these particular examples the cæcum itself was also quite normal in appearance.

Further, it may be said that when a perforation of the cæcum is discovered at the bottom of a perityphlitic abscess, it affords no evidence that the mischief began in the cæcum, or that the perforation was primary. Most of the perforations so met with are secondary, the trouble having begun in the appendix, and the pus having made its way into the cæcum from without.

Finally, it must be allowed that the appendix is frequently infected from a diseased cæcum.

B. Perityphlitis taking origin from the appendix.—(*a*) *With catarrh or ulceration of the appendix.*—Catarrh of the appendix is evidently of very common occurrence. It is quite commonly found in post-mortem examinations by those who are taking note especially of the condition of the appendix. It may be safe to conclude that simple and uncomplicated catarrh of the appendix leads to no symptoms, and cannot be clinically recognised. There is evidence that it may be acute or chronic, and that it may be entirely recovered from. Its origin is due, no doubt, to the same causes which lead to catarrh in other parts of the intestine. Yet now and then it appears to be induced by a minute fragment of fæcal matter retained in the appendix which, as a solid and decomposing foreign body, sets up irritation. Very often, indeed, I believe the catarrh extends to the appendix from the cæcum. It is common to observe that mischief in the appendix, leading to perityphlitis, is set up by constipation, by a lodgment of fæcal matter, or of a mass of undigested food in the cæcum. It is reasonable to conclude that some catarrh or even ulceration of the cæcum may be induced by such accumulation, and that the inflammation may spread to the appendix, which seems to be peculiarly susceptible to catarrh.

In a few instances a minute foreign body is the cause of the catarrh. Such instances will be alluded to later.

The morbid changes produced by the catarrh are precisely the same as in other parts of the intestinal canal. The epithelium of the surface, as well as that which lines the crypts of Lieberkuhn, is shed, the retiform tissue becomes infiltrated with leucocytes, and the whole mucous membrane is thickened. The process may end in complete recovery, no doubt; but, if long continued, it may lead to one or other of the following results:—

(*a*) The watery discharge, produced by long-continued catarrh, may lead to an inorganic *concretion*. These concretions are no doubt formed from the salts contained in the catarrhal fluid. A precisely similar formation of a stony concretion, as a result of long-continued catarrh, is met with in the nose as a rhinolith. The rhinolith appears in all cases to be the product of long-continued nasal catarrh. Occasionally a minute speck of foreign matter, such as a piece of nutshell, forms the nucleus of the concretion. The concretions are usually of a mortary consistence moderately friable, and are composed of phosphates of lime and magnesia and of carbonate of lime, mixed or stained with a little fæcal matter. Some have been found to contain cholesterin. Owing to the slight admixture of fæcal matter they are often called fæcal concretions. I do not know that the common assertion that they contain "inspissated mucus" is well founded. Some of them are veritable stony concretions, and can be evenly divided with a fine saw. The firmer stones are much like the rhinoliths on section. I have never satisfied myself that, as some affirm, the actual nucleus of these concretions is composed of fæcal

matter. I have found bodies in the appendix with a putty-like interior, resembling fæcal matter with a casing of lime salts. The real concretions must be clearly distinguished from the little pellets of moulded and often hard and brittle fæcal matter which are often met with in the appendix.

The real concretions vary greatly in size. They are generally of even and symmetrical outline. Some are very minute, others are over an inch long. The largest which I have myself removed was more than an inch in length and nearly half an inch in diameter. Blackadder found an earthy-looking concretion, the size of a thrush's egg, in a man's appendix. It had caused no trouble during life.

These concretions often bear the very closest resemblance to certain vegetable products. The smallest are readily mistaken for tomato-seeds and fig-seeds, the larger for orange-pips, grape-seeds, or grains of wheat, and the largest for cherry-stones and date-stones. I possess two concretions which, before section, I could not tell from a cherry-stone and a date-stone respectively. I have never found such seeds or fruit-stones in the appendix, and I believe them to be exceedingly rare. It is impossible to understand how a cherry-stone or date-stone, or even an orange-pip, could enter the appendix, and the imitation made by a concretion is so exact, that I am disposed to think that very many of the reported cases of foreign bodies are examples of concretions. That a grain of wheat could reach the appendix quite unchanged is most difficult to believe. A portion of a grape-skin is very remarkably imitated by a layer of thick, glutinous, dark greenish-purple mucus.

The frequency with which concretions are found in the appendix in cases of typhlitis is variously stated by different authors. Matterstock found them in 53 of the collected cases, Fitz in 47, Krafft in 34, and Murphy in 30. I am inclined to think that the lower estimates, that is, about 30 per cent, are more near the truth. In 400 post-mortem examinations of all kinds, Ribbert found a fæcal concretion in the appendix in 10 per cent of the bodies.

(β) In the second place, long-continued catarrh may lead to great *thickening of the appendix.* The tube is found to be of unusual size and stiff and straight. Its wall may be from $\frac{1}{6}$ to $\frac{1}{8}$ of an inch in thickness, and its lumen may be found full of mucus.

(γ) Thirdly, catarrh may lead, and apparently often does lead, to *ulceration of the appendix.* Some degree of ulceration of this little process is very common in perityphlitis.

The ulcer need not be preceded by definite catarrh, it may be primary. The primary ulcer may be due to the presence of a foreign body, to septic infection, to typhoid ulceration, or to tuberculosis. Foreign bodies are not common in the appendix, and but very rarely are they the cause of perityphlitis. Many reputed foreign bodies, as already stated, are really concretions formed in the appendix itself as a result of chronic catarrh. Among the foreign substances which have been actually found in the vermiform process are the following: pins, minute fragments of bone and of nutshell, small shot, bristles, splinters

me to think that 5 per cent is more nearly correct [illegible]

In some instances the foreign bodies may escape [illegible] through the ulcerous openings they may make in the [illegible]

[illegible] have been found very often in association [illegible] the condition having been preceded by some [illegible]

The aspect of the ulcer in the appendix [illegible] There may be several ulcers of the [illegible] be [illegible]. If perforation take place [illegible] spot. When the ulceration has been [illegible] is found to be much thickened and its [illegible]

One must assume that certain ulcer [illegible] due to an acute septic infection, with or without [illegible] manner in which that infection was brought about.

Typhoid [illegible] are rare in the appendix [illegible] cases of typhoid fever which I have seen [illegible] [illegible]

[illegible]

This perforation may be of any size or shape, from a mere pinhole to what appears to be a rent of a greater part of the limb. The perforation may take place into a normal peritoneal cavity, or into a space more or less confined by adhesions.

It is by no means necessary that an ulcer of the appendix should lead to perforation in order to produce the phenomena of perityphlitis. Over and over again the vermiform process, removed from the midst of a perityphlitic abscess, has revealed no evidence of perforation. If all the cases of perityphlitis of all varieties and degrees be considered together, then it may be said that in the majority there is no perforation: on the other hand, it is estimated that in from 60 to 70 per cent of the really acute cases a perforation of the process may be expected.

In the second place, the ulcer of the appendix may lead to *stricture*, or to more or less obliteration of the lumen of the process. The mucous membrane over a varying extent is destroyed by the ulcerative process; granulation tissue covers the denuded surface, and, if the two opposed walls of the little tube adhere, a stricture results. The whole of the lumen of the process may be occupied by granulation tissue, and several strictures may form in the same appendix. The stricture and its results are dealt with on page 888. In not a few instances, where the ulceration occupies the whole internal surface of the appendix, the lumen of the tube finally becomes entirely obliterated, fibrous tissue occupies the once free passage from one end to the other, and the appendix is converted into a solid fibrous cord. This is one of the natural processes of cure in perityphlitis. Any degree of stenosis may result from this condition; there may be a mere linear stricture constricting the appendix like a fine ring; there may be several strictures of like character, or the appendix for one-fourth, or one-third, or one-half of its length may be converted into a fibrous cord.

Between multiple strictures cavities of varying dimensions may be left. The whole lumen of the process, although patent, may be found evenly narrowed from one end to the other.

Now and then the obliteration of the appendix is so complete, that only a careful examination of the adhesions which probably exist around the cæcum will reveal any trace of the organ. Ribbert found in 400 post-mortems more or less obliteration of the appendix in 23 per cent, and found also—as may have been expected—that this particular lesion was more common as age advanced.

(*b*) *With gangrene of the appendix.*—In some cases the appendix is the seat of a more or less extensive sloughing or gangrenous process. Clinically this condition is nearly always associated with symptoms of a very severe and acute character, especially when the gangrene is complete.

There is evidence to show that the whole of the part may become gangrenous in twenty-four hours. The degree of the sloughing varies; in some instances the whole of the mucous membrane becomes necrosed, or there is considerable sloughing of the membrane in patches; in other

examples the whole of the appendix may be gangrenous from base to tip. It may, indeed, be found detached as a slough, and I have so discovered it floating in a perityphlitic abscess. Or the gangrene may be of much more limited extent; the tip only of the process may have perished, or, curiously enough, only the base may have sloughed. In the latter instance a more or less sound appendix may be found detached from the cæcum by one of these basal sloughs. The gangrene may involve a portion only of the appendix wall, and in some cases the limited extent of it may be inexplicable. The causes of gangrene of the vermiform process are not always apparent, but it is safe to conclude that they are the same as those which produce gangrene elsewhere. In not a few instances the sloughing appears to be the result of a septic infection of an already damaged appendix. In some cases, for instance, in which the whole organ had perished, a concretion has been found. In such conditions it would appear as if a virulent septic inflammation had made its appearance in an appendix long diseased. Certain it is, that a concretion may exist in the appendix for years and cause no trouble.

In another series of instances there is a suggestion that the convoluted appendix may have become so twisted upon itself as to have brought about an occlusion of its vessels. A like condition is seen in volvulus of a loop of bowel like the sigmoid flexure. Extreme alterations in the volume of the cæcum may lead to such a twisting.

In certain examples of gangrene it is possible that the vessels leading to the appendix may have become thrombosed. I have found these vessels very nearly occluded in some examples of relapsing perityphlitis. Mere bending of the appendix mesentery, or the pressure of adhesions, may lead to such a diminution in the lumen of the vessels of the process. Foreign bodies have been credited with the production of gangrene, but upon no substantial evidence. On the whole it is probable that most of the examples of extensive gangrene are due to infection of the appendix with pyogenetic micro-organisms such as the streptococci.

(*c*) *With stricture or occlusion of the appendix.*—Stricture of the appendix plays a prominent part in the production of perityphlitis, and especially of the relapsing form. The mode of production of these strictures has been already alluded to (page 887).

The effect they produce varies according to the condition of the mucous membrane on the distal side of the stenosed part. If this mucous membrane be quite healthy, the distal part of the appendix becomes distended with mucus. The cyst thus produced has a very remarkable appearance; it is usually of considerable size, is sausage-shaped, is of even outline, and the proximal end of it may bulge into the cæcum as a large hemispherical swelling. The cyst contains from one to four ounces of perfectly white, translucent, odourless mucus. An excellent specimen of such a cyst is to be found in the Cambridge Museum; it was removed from an old subject during a post-mortem examination. This cystic dilatation has been termed a "mucocele" by Féré. Weir describes a case, and compares the contents to the vitreous humour. One

of these translucent cysts is figured in Coats' *Manual of Pathology*, and Dr. Wilks met with another example. In no case have any adhesions been found associated with these cysts. Porter describes two examples in which the mucous membrane was quite healthy, but in which the other coats were the seat of some chronic inflammatory thickening. The condition is probably identical with Virchow's "colloid degeneration of the appendix." It is said that this curious cyst does not give rise to any symptoms, so that in nearly all instances it was only discovered on post-mortem examination. The occlusion appears always to be at the cæcal end of the appendix, and it may be due to a congenital defect. I have met with one instance during life. The patient, a man of thirty, had always been the subject of constipation. Since the age of eighteen he had suffered at intervals from a vague and not severe degree of colic, described as "congested liver." He had had also a definite attack of gastritis. For some months before I saw him he had had a "heavy pain" in the right iliac region; it was worse on exertion, and extended down the right thigh and into the right testicle. It was never severe. He complained so much of this "heavy feeling," that I opened the abdomen and discovered in the pelvis the cæcum and a cystic appendix. There were no adhesions. The appendix was shaped like a banana, and measured $4\frac{3}{4}$ inches in length by $1\frac{1}{2}$ inch in width. It was elastic, smooth, and quite translucent. On removal a quantity of clear, white, perfectly translucent mucus escaped from it, very much like the material which is met with in a ganglion. After the operation the symptoms vanished.

If the mucous membrane beyond the strictured spot be unhealthy—as it usually is—then the distal part of the appendix is found to be distended with muco-pus, or pus, or opaque and ill-looking and often ill-smelling mucus. These cysts may be oval, cylindrical, or round; they are usually surrounded by adhesions of varying density. The cyst wall is as a rule very thick, and the swelling may be so tense as to feel—when exposed by operation—like a stone. In size these cystic swellings are seldom large; one of the size of a walnut would be quite large: I have encountered some of considerable size which have measured as much as $1\frac{1}{2}$ inch across, but these are rare. In these cysts the mucous membrane is found to have been destroyed by ulceration. The cysts are apt to perforate and to burst; the perforation leads to an attack of perityphlitis, and the contents escape more or less completely from the appendix. If the patient recovers the perforation heals, and in due course the cyst fills up again, and bursts again; thus the conditions for repeated relapses are produced.

Sometimes the cyst ruptures, and a large rent is produced. These cases usually lead to an abscess, and if the abscess cavity heal, the patient is cured, as the appendix becomes practically obliterated. Some of the fibrous cords which are found in the place of the appendix are no doubt the result of the process here described.

It is not necessary, however, that a stricture should exist in order to

produce a cystic dilatation of the appendix. The little tube may b occluded by acute bending upon itself, or by torsion around the line o its long axis. The process so deformed is held in position by adhesions and by freeing the adhesions the lumen can be restored.

As in the case of the stricture, muco-pus or, at any rate, very u healthy-looking mucus must inevitably accumulate in the appendix beyond the stenosed part. The cysts produced by such accumulation are, as a rule, larger and more globular than those produced by actual stricture. I think that in the cases of acute bending or torsion the mucous membran is probably more healthy than in the cases associated with stricture. I have met with cysts measuring 2 inches across, and on dividing th adhesions which bound down the appendix have seen such cysts empt themselves entirely. In some of these cases the little tube has bee so bent upon itself as to be in the form of an acutely bent hoo In instances in which there was torsion, so slight a disturbance o the adhesions allowed the cyst to empty itself that I imagine su emptying must take place from time to time during life. It must b emphasised that in these examples there is no real stricture. The cyst part of the appendix may, however, give way by perforation, or by more extensive bursting; but such an accident is less common than the cases with stricture.

The subject of stricture and occlusion of the appendix is alluded again in dealing with relapsing perityphlitis (page 918).

(*d*) *With actinomycosis of the appendix.*—A few examples of th rare condition of the appendix have been recorded. One of the mo complete, as well as the first described, is recorded by Dr. Ransom (s page 917). The patient in this instance was a man aged fifty, in whc right iliac region a painful and tender swelling appeared. An absc formed in due course and discharged through the skin. In the pus clum of the actinomyces were found. The patient died. At the necrop the abdomen was found free from general peritonitis. The vermifo appendix was contorted, and was almost buried in dense adhesic and thickened peritoneum. The cæcal aperture of the appendix w patent. The tube itself was in part dilated; its walls were irregul and in several places communications led to an extensive abscess behi the cæcum. The cæcum was free from evident disease. The liver co tained a large focus of pus, in which colonies of the ray fungus w found. Microscopic examination of the retro-cæcal abscess and of t appendix where it communicated with the abscess failed to reveal a colonies of the actinomyces. Dr. Ransom was of opinion that t appendix was the primary seat of infection, and suggested that a fragm of corn or grass bearing the parasite might have lodged in the vermifo process. (See also a case by Otto Lanz. *Vide* art. "Actinomycosi vol. ii. p. 81.)

C. The peritonitis.—The peritonitis, which is the basis of the affecti under discussion, may be of any degree and of any extent. It may general or local, suppurative or non-suppurative; or a localised s

purative peritonitis may be associated with a diffused non-suppurative form of peritoneal inflammation.

In the majority of the cases the peritonitis is entirely localised, and—in a less large proportion—it does not pass on to suppuration. The peritonitis is evidently due in all instances to the escape of bacteria-laden material from the appendix, or, in a few examples, from the cæcum. It would appear that the micro-organism which is most active in the production of this particular form of peritonitis is the bacillus coli communis. Tavel and Lanz examined twenty-three cases of disease in the appendix. The examination was made either of the pus within or without the appendix, or of the exudation into the peritoneal cavity immediately around the diseased process. The material for this bacteriological investigation was obtained during the life of the patients. In three no micro-organisms were found; in nineteen the colon bacillus was found either alone or in association with other pathogenetic germs. The streptococcus was found in several cases; the pneumococcus was met with in two, but not in pure culture. The staphylococcus pyogenes citreus was in one instance the only micro-organism discovered. Hodenpyl deals with twenty-seven cases of perityphlitis. In twenty-five the bacillus coli communis was the only micro-organism found. In one case this bacillus was found in association with streptococci, and in the remaining case the streptococcus was found alone.

Many other investigators have demonstrated the same facts. I may say that the interior of the appendix of the fœtus at birth has been shown to be free from micro-organisms. In the adult the streptococcus has been often found in the healthy appendix.

The bacillus coli communis exists in the normally human body, and is said to be the most abundant and the most constant of the bacteria found in the healthy man. Its natural habitat is the bowel, and it has been demonstrated along the whole length of the alimentary canal from the mouth to the anus. The micro-organism has this notable feature, that it varies greatly in its virulence. So far as experiments upon animals are of value, it would appear to be harmless when taken from the normal intestine. If, however, the bowel become the seat of almost any morbid change the bacillus becomes virulent at once. Virulence has been found in cases in which the bowel was obstructed, strangulated, or inflamed; in congestion of the intestine; in diarrhœa; in advanced constipation, and in other abnormal states. This point is of no little moment in the etiology of perityphlitis. In a large number of cases the attack is preceded by constipation, by the decomposition of undigested matters in the bowel, by diarrhœa, by exposure to cold, or possibly, although rarely, by injury. If measures could be adopted to render the bacterium non-virulent, then a great step would have been taken in the preventive treatment of this disease.

Two factors, at least, appear to be necessary to produce an attack of localised peritonitis through the medium of the appendix. The first is such a condition within the bowel as will render the colon bacillus

virulent; and the second is such a lesion in the appendix as will perm that bacillus to reach the peritoneum. The invasion of the peritoneu by the micro-organism gives rise to sudden, violent, and acute symptoms to an outbreak which is often so abrupt and intense as to produce th impression that the appendix has become perforated. It is not necessar however, that there should be a definite and demonstrable perforation t account for acute and sudden outbreaks of perityphlitis; in not a few c such attacks I have failed—on exposing the appendix by operation—t detect any obvious perforation.

The character and extent of the peritonitis produced will depen obviously upon the "dose" of septic material which enters the pe toneal cavity, and also upon the manner of its entrance. We must no whether the diseased appendix lay perfectly free in a normal sero cavity, or whether it was located in a pouch, or lodged in the pelvis, surrounded by adhesions, or buried in a mass of attached omentum, fixed to the wall of the iliac fossa. The effusion produced may be scan or copious, serous, sero-fibrinous, or sero-purulent, or wholly purulent. may be free from smell, but in the majority of instances it has a mo offensive and often fæcal odour. The least severe cases are usual associated with a little clear serous lemon-coloured fluid; the most seve are usually associated with foul pus, but in some of the fatal cases in t place of pus a thin greenish-gray fluid of an intensely offensive and ac odour is seen. This I have found even at a comparatively late peri in the case, and in instances as a rule in which general symptoms septic poisoning were prominent. This greenish exudation is at fi clear and then becomes semi-opaque. It is not large in amount.

In chronic cases a yellowish-white, soft custard-like material is som times produced, which is often met with in operations upon the appen during a quiescent period; I believe it to be neither changed pus n a residuum left by pus. In some cases I have scraped away a f teaspoonful of this matter from the appendix and its neighbourhood.

Attention may here be drawn to the curious uncertainty of the s purative process in perityphlitis. Pus may be present in forty-eig hours, and, according to some, even at an earlier period. On the oth hand, the case may progress for two or three weeks with persisti evidences of acute inflammation, and possibly with short periods apparent improvement, but with no local evidences of pus. At the e of these two, three, and even four weeks pus may make itself evident.

Inasmuch as the peritonitis in perityphlitis is usually localised follows that adhesions are the rule in this affection, and indeed are abs in the most acute and rapidly fatal cases only.

D. The abscess.—The abscess is in reality a collection of matter c to localised suppurative peritonitis. It is in the peritoneal cavity, l only in a limited part of it, being isolated by adhesions from the gr serous space. These adhesions are formed to a large extent by adher coils of small intestine and by attached omentum. Those who adv the very dangerous process of scraping the walls of these abscesses shou

remember that these walls on one side, at least, are made up of loops of intestine which have become glued together. It is necessary to repeat what has been already pointed out, namely, that an abscess round the appendix does not of necessity imply a visible perforation of that little tube; it is merely an evidence that noxious micro-organisms have found their way from the interior of the appendix to the serous membrane which covers it. Abscesses have been found around appendixes which not only are not visibly perforated, but which contain no pus, and are simply in a state of chronic catarrh.

The material in the abscess varies. There may be merely a scanty, thin, turbid, greenish liquid with a very ill odour. There may be a sero-purulent exudation. There may be laudable pus. The pus has usually an offensive smell; it may have an odour actually fæcal, or it may contain fæcal matter; but the last-named condition is uncommon. The most usual matter evacuated from the abscess is very ill-smelling pus of a "dirty" colour, somewhat thin, and often mixed with clots of fibrinous matter.

The quantity of pus found in the abscess varies. It is usually some three or four ounces; but it may contain some pints. In one case which had been long neglected I evacuated an abscess which contained two quarts.

The abscess cavity is apt to contain gas, the result probably of decomposition. In rare instances the gas is excessive in amount, and the swelling caused by the abscess may be as resonant as the surrounding intestines. Morton reports a case of a perityphlitic abscess from which on puncture gas only escaped, but a deeper puncture revealed pus. In one case, in my own practice, I cut down upon an iliac abscess following upon perityphlitis, and opened a large cavity containing gas, and presenting in its depths so very small a quantity of pus that I could not help thinking that the matter might have escaped by an opening into the bowel; there was no evidence, however, of such an opening, and the cavity healed rapidly and well.

In a case reported in the *Med. News* (U.S.A., December 17, 1892) the abscess cavity contained not only gas and pus but some fæcal matter also.

The relation between the size of the swelling found on the right iliac fossa and the amount of pus which is discovered in the abscess is not always direct; the swelling may be extensive, and yet the amount of pus small. The swelling is made up of the cæcum and sundry coils of small intestine which are adherent, and the walls of which are congested and œdematous. Between and around the coils are much adherent lymph, much effusion, and most probably a considerable mass of thickened and adherent omentum; in not a few instances there is also a collection of fæcal matter in the cæcum. The swelling is apparently increased again by swelling in the protecting parietes, and by contraction of the muscle over it.

In the majority of abscesses which are evacuated by operation the

appendix does not come into view; it is obscured and indeed buried by recent adhesions. It may, however, present itself and be found perforated or gangrenous, as a whole or in part. It may be found actually detached—as a result of gangrene of its base—and lying free in the abscess cavity. Calcareous or fæcal concretions or, in rare instances, foreign bodies may be found in the abscess cavity, such substances having escaped through breaches in the appendix wall produced by sloughing.

A perforation may be found in the cæcum. In a few rare instances this may be primary, and due to mischief starting in the wall of the cæcum, the appendix itself being sound. In the great majority of the examples, however, the perforation is secondary and is due to the abscess —which has originated in the appendix—making its way into the cæcum. The perforation under such circumstances takes place from without. In one case I met with three separate perforations of this character in the cæcum.

The perityphlitic abscess is as a rule very extensively and deeply loculated, and may even appear to be made up of several distinct collections of pus. This disposition to the formation of loculi or pockets often causes much difficulty in the healing. The secondary pouches may extend between coils of bowel, or make their way into the pelvis or into the loin.

As regards situation the perityphlitic abscess, in the great majority of instances, is situated in the right iliac fossa, but it may be found entirely limited to the pelvis on the one hand, or to the lumbar region on the other; the iliac fossa in either case being quite free. Such unusual positions are determined by unusual positions of the appendix. Fowler gives three cases in which the abscess was to the left of the median line; in one example the cæcum lay on the promontory of the sacrum, and in the other two was beneath the umbilicus. Dr. M'Callum, in a post-mortem examination of the body of a woman aged thirty, found the appendix adherent to the upper surface of the liver on the right of the gall-bladder. The appendix was obliterated by disease. There had been an abscess around it which had made its way through the lung, and had finally discharged itself through the eighth intercostal space. The abscess had healed. I have opened by operation a subhepatic abscess due to disease of an appendix attached to an undescended cæcum. In this instance the right iliac fossa was entirely free from any signs of inflammation.

The perityphlitic abscess, although primarily intraperitoneal, may make its way into the subperitoneal connective tissue; such an invasion, however, is not common. The extraperitoneal abscess, when it exists, usually occupies the lumbar region, and may extend very widely in the lax tissue of that district. I have seen such an abscess fill up the whole of the region between the lower ribs and the iliac crest.

The extraperitoneal abscess may be found on the superficial aspect of the psoas muscle, and lie distinctly behind the serous membrane

It may actually open up the sheath of the psoas muscle, and extend towards the spine in the substance of that muscle. Such cases have been well illustrated by Dr. Kelynack.

It is not at all uncommon to find that the abscess—in a chronic case—has extended upwards behind the ascending colon; such an abscess may reach the diaphragm, may perforate it, and empty itself into the pleura or the lung. It is possible that certain of these abscesses are extraperitoneal, but I believe that in the majority of cases they are still intraperitoneal, and that they follow a course which is bounded by peritoneal adhesions.

Finally, it remains to consider in which direction the abscess will burst, supposing it not to be evacuated by the surgeon. Bull, in an analysis of 57 cases, gives the following as the result of an inquiry into this matter:—In 28 examples the abscess burst through the anterior abdominal parietes, in 15 it entered the cæcum, and in 8 the general peritoneal cavity. In the remaining 6 examples the abscess entered the thorax, rectum, and bladder, in 2 cases of each respectively. The pus may pass down the thigh, and very closely imitate a psoas abscess. It may extend in front of the hip-joint and produce symptoms akin to those of hip disease; indeed I have seen a case in a boy in which this diagnosis had been made. The pus has even made its way into the hip-joint, and in other instances has laid bare the bone of the iliac fossa. It has often made its way into the pelvis, and has produced some of the varied and manifold symptoms of pelvic abscess. Again, it has passed down a hernial sac, and made its escape through the skin of the scrotum.

ETIOLOGY.—Some features in the etiology of perityphlitis have been already indicated in the preceding section. Perityphlitis represents the very commonest variety of peritonitis, and the remarkable frequency of inflammation in this position is due to the fact that the appendix is one of the weakest points within the abdomen. It has been estimated by pathologists that in 20 per cent of the bodies examined in the post-mortem room some evidence of disease of the appendix will be found. Certainly the same cannot be said of any other organ within the abdomen.

Inheritance.—Heredity is not easy to demonstrate in a disease so common as that under consideration, but it is impossible not to be struck with the fact that perityphlitis runs often in families. I have met with a family among whose members were five subjects of perityphlitis. I have also noticed that the association of this affection in families is not so often that of parent and child as of brother and sister. This has seemed to me to be the most common coincidence.

Age and Sex.—Perityphlitis may be met with in patients of all ages. Tenger reports a case in an infant aged seven weeks. It led to a fatal peritonitis. Cases have been recorded in children respectively seven and thirteen months of age. On the other hand, the disease has been met with in patients of advanced age; one of my own patients

was an old edentulous man who had his first attack at the age of seven two. Several cases have been recorded in still older persons. Th greater proportion of the cases occur in young adults, and the period o greatest frequency is between ten and twenty.

The following table is compiled from the cases collected by D Hawkins (224 cases from St. Thomas's Hospital) and by Dr. Fitz (22 cases from general sources), the total being 452 cases :—

Age.	Proportion of cases. Per cent.
5 to 10 years	10·8
10 to 20 years	40·7
20 to 30 years	29·0
30 to 40 years	11·5
40 to 50 years	4·6
Over 50 years	3·4
	100·0

In the matter of sex the trouble is very much more common men than in women, the proportion in the two sexes being about to 22. Some writers place the relation as high as 80 men to 20 wom in every 100 cases.

Season.—Hospital statistics show that attacks of perityphlitis a more common in the summer than in the winter. Dr. Hawkins, analysing 224 cases of perityphlitis treated in St. Thomas's Hospit notes that 134 came under notice in the six summer months, and 90 the six winter months.

Injury.—Dr. Fitz, as a result of the examination of the large series cases he had collected, considered that an injury was the cause of attack in 10 per cent of the examples. This percentage is, I thi somewhat too high. It is not uncommon, however, to find instances which an attack of perityphlitis has apparently followed directly up some violence. Many cases are ascribed to a strain, to violent exerti and to a blow. In one case under my care the symptoms immediat followed a blow on the abdomen; in another case acute sympto began abruptly while the patient was straining to support a hea weight. It is probable that in these instances the appendix was alrea diseased, and that the violence merely caused a greatly attenuat appendix wall to give way. Some of the cases in which a blow appeared to cause an attack have been very acute, but this has b by no means invariably the case. I am not aware that exposure to c has been clearly shown to have been an exciting cause of perityphlitis.

Condition of the alimentary canal.—An examination of cases fr a clinical standpoint makes it evident that the commonest exciting ca of perityphlitis is the lodgment of undigested matter or of fæcal masses the colon. Mere constipation alone would not appear to be sufficient. is generally allowed that constipation is more frequent among wom than among men, and yet, as we have seen, perityphlitis is nearly f times more common in men than in women.

Patients who have had repeated attacks of perityphlitis often say that so long as they keep their bowels acting they can avoid an attack, but if they allow an accumulation to take place an attack may be expected. There is strong evidence to support the belief that masses of undigested food in the cæcum are much more active exciting causes of this trouble than are mere collections of what may be termed normal fæcal matter.

In this connection two facts have to be specially noticed.

(*a*) In the first place, defective masticating teeth are exceedingly common among the subjects of this disease, and especially among those who have passed the period of youth. Over and over again it would appear as if the want of proper and efficient teeth had been the direct cause of the attacks. Such patients often bolt their food, and such meat as they eat can hardly reach the stomach in a condition fitted for complete digestion. The bolus passes into the bowel still ill digested; it fails to stimulate normal peristalsis; it is prone to lodge in the great receptacle, the cæcum, where it decomposes and, if there be any existing lesion of the appendix, must tend to encourage the morbid change.

A large proportion of the subjects of perityphlitis—especially the subjects above the age of twenty—are chronic dyspeptics. They bolt their food, or their meals are irregular, or they hurry over their meals, or they begin active work as soon as the repast is finished. Many of these people have a morbid appetite; many of them are aware that an attack is generally preceded by an aggravation of the dyspeptic symptoms. One medical man, who had had many attacks of perityphlitis, informed me that he was often warned of an attack by the passage of exceptionally offensive flatus for a day or more previously. Another patient had learned that some little while before his attacks—which were of sudden onset—his urine was very thick and high-coloured.

Commercial travellers, who are in incessant movement, and whose meals are irregular, are often the subjects of perityphlitis. In other persons a first attack has occurred in connection with hurried travelling, as during a hasty Continental holiday. Indeed, the holiday season is generally very productive of cases of perityphlitis.

Certain of these patients are nervous persons, who seem to be victims of a kind of nervous dyspepsia. In an isolated case or two it would appear as if the dyspepsia which preceded the perityphlitis had been induced, or at least fostered, by excessive smoking after meals.

(*b*) In the second place, the history of an indigestible meal is comparatively frequent in the account of events which immediately preceded the actual attack.

Among the articles of diet which have been conspicuous in this respect may be mentioned nuts, pine-apple, coarse vegetables such as turnips and cabbage, cheese, mushrooms, mussels, haricot beans, cucumber, lobster, and so forth. When the bowel is evacuated in the course of an attack of perityphlitis, the offending substance can often be detected. In one case (in a young girl), remarkable masses, made up of partly masti-

cated nuts, were passed for several days, during the progress of the attack. One patient was convinced that he could produce an attack by eating eggs. Many patients—especially those at or past middle life—have apparently owed an attack to the consumption of an immoderate quantity of milk.

In a very small proportion of cases diarrhœa would seem to have been the exciting cause of an attack.

In a larger proportion of cases the attack is preceded by colitis, marked by frequent loose motions, and the passage of much mucus and intestinal casts. Not infrequently this colitis has been excited by a fæcal accumulation in the colon.

It has been assumed, in some instances, that a purgative has induced an attack, but more probably the actual cause was the condition which called for the aperient. I have noticed that in young women an attack is not infrequently associated with the appearance of menstruation. In one case under my care four attacks out of six were thus associated.

Foreign substances.—The part played by fæcal and calcareous concretions, and by actual foreign bodies which have been swallowed, has been already fully discussed (p. 884). Talamon states that, in the fatal cases, a concretion of some kind, or a foreign body, is found in 60 per cent of the bodies examined.

As already pointed out, the intrusion of foreign bodies is by no means so common as was at one time supposed.

Constitutional conditions.—*Gout.*—Dr. Alexander Haig believes "that gouty perityphlitis is extremely common, or, in other words, that nearly all perityphlitis owns this origin; and that, if all cases were at once treated by salicylates, further troubles requiring surgical procedures would be rare." Dr. Haig cites several cases in which the peritonitis yielded to salicylates, and it would appear that the diagnosis of gouty perityphlitis is mainly based upon the circumstance that relief is afforded by the salicylates. Dr. Haig's views need confirmation, and are not in accord with the common experience of medical men.

Rheumatism.—Dr. Burney Yeo records a case (in a girl of eighteen) of symptoms of perityphlitis, associated with pains in the arms and knees. The patient had had acute rheumatism. The temperature fell and the symptoms vanished under the influence of sodium salicylate. The temperature reached 105°, the patient was very constipated, and the symptoms of perityphlitis appeared before the joint phenomena. Dr. Yeo would seem to regard the disappearance of the symptoms, when the salicylates were administered, as evidence of the rheumatic nature of the affection. There is little evidence to support the assertion that rheumatic peritonitis exists (*vide* p. 633), and the nature of the alleged changes in the cæcum and appendix in this disorder have, I believe, never been defined.

Perityphlitis due to typhoid ulceration, tuberculous ulceration, dysentery, actinomycosis, and epithelioma, has been already described.

Influenza and pneumonia have been supposed by some observers to be exciting causes of perityphlitis, but no definite connection has been made

manifest, and the matter rests, I believe, upon no more substantial basis than the clinical fact that perityphlitis has appeared in patients convalescent from influenza or pneumonia.

SYMPTOMS.—The clinical manifestations of perityphlitis are extremely various, and differ remarkably in degree and duration. The attack may assume any grade, from a sudden and intensely acute diffused peritonitis, which is rapidly fatal, to an attack marked by slight pain and tenderness in the right iliac fossa, with possibly nausea or vomiting, and ending in recovery in some twenty-four hours or even less.

On the one hand, an attack which begins violently may soon subside and end favourably; on the other hand, an attack which begins mildly, and which follows a moderate course for a few days, may then develop very acute symptoms, and end fatally.

It is by no means possible to distinguish the cases which will end in suppuration from those which will not. Pus has been discovered as early as forty-eight hours from the onset of the attack; in other cases it has not been manifest until the disease has been in existence for two or even more weeks. In one case under my care the temperature remained high (100° to 102°, and at first to 103°) for twenty-two days. At the end of that time the iliac swelling was incised. No pus was discovered, and the patient made a perfect recovery. It is of little service to attempt to divide the cases clinically into those which suppurate and those which do not, for the distinguishing features which should be present before the abscess is in evidence, or the case has ended favourably, are not definite enough to warrant such a classification. Some authors have divided the cases clinically into those associated with local peritonitis and those marked by general peritonitis. In the former category they have placed what may be called the average cases, and in the latter the cases which are very acute and for the most part fatal. This classification is, however, not entirely satisfactory.

The peritonitis may be local at first, and not become general until a week or even more has elapsed; and if the cases placed in this category are restricted to those in which the peritonitis is general from the onset, then the arrangement is of necessity very imperfect.

Classification.—It is not possible, indeed, to found a satisfactory clinical classification of perityphlitis upon purely pathological bases. The clinical phenomena cannot provide a sure forecast of the condition to be revealed on operation or necropsy. It is probably better to adopt some arbitrary division of the cases such as would be suggested if the clinical manifestations were regarded alone. The following classification, framed on these lines, is convenient for purposes of description:—I. An ordinary attack, the case ending in resolution; II. The case ending in suppuration; III. An attack of the mildest type; IV. A most intense and acute attack; and V. Certain peculiar forms of perityphlitis.

Relative frequency of the different varieties.—The cases dealt with under the third heading do not come—except rarely—under treat-

ment in hospitals, and cannot therefore be treated statistically. Indeed, this class may be considered as unrepresented in hospital records. It is often conspicuous, however, in the history of relapsing perityphlitis. The cases under the fourth heading are usually attended by general peritonitis. Dr. Hawkins, in dealing with 264 cases of perityphlitis treated at St. Thomas's Hospital, divides them into three divisions—i. Those attended by non-suppurative local peritonitis; ii. those associated with suppurative localised peritonitis; and iii. those attended by general peritonitis. The 264 cases are distributed as follows:—

	No. of Cases.	Deaths.	Mortality. Per cent.
i. Non-suppurative local peritonitis .	190	...	...
ii. Suppurative local peritonitis . .	38	10	26·3
iii. General peritonitis	36	27	75·0
	264	37	...

General mortality, 14 per cent.

I. An ordinary attack ending in resolution.—The *general features* of an attack are sudden severe abdominal pain more or less definitely located in the right iliac fossa, nausea or vomiting, constipation, rise of temperature, the development of a tender area in the cæcal region, and the formation of a tumour which is very tender, and usually dull on percussion. There is some distension of the abdomen and some rigidity of its walls. The pulse may be thready, and the tongue is furred. There is much prostration. The fever subsides in about a week, and within seven to fourteen days all the acute symptoms have passed away. The *onset* is usually sudden. The attack often comes on at night or in the early morning. In some cases of relapsing perityphlitis the attack sets in at about the same time on many successive occasions. An initial rigor may be said to be very rare, and no clinical or pathological importance is to be attached to the symptom. The patient may be in perfect health at the time of the attack, or the onset of acute symptoms may have been preceded by more or less abdominal uneasiness, by flatulence or colic with a sense of distension, by diarrhœa, or by troublesome constipation. In many cases there had been undoubted dyspepsia or a "liver attack" before the onset of the symptoms; there was a nasty taste in the mouth, the urine was thick, or the patient had felt vaguely out of sorts.

The first symptom is abdominal *pain*. In about 70 per cent of the cases this pain is localised at first just above the umbilicus, or across the belly at the level of that point. The position indicated is about that of the superior mesenteric plexus. In the remaining 30 per cent of the cases the pain is from the first definitely localised

in the right iliac fossa. Should the pain be, first of all, complained of about the umbilicus, almost invariably it becomes definitely felt in the right iliac fossa in twelve to thirty hours. It is in first attacks especially that the pain is localised about the umbilicus, and in some instances it remains so located throughout the attack. I have seen cases in which, during the first two attacks, the pain was complained of only in the upper segment of the abdomen. After the patient has had one or two attacks the pain in subsequent outbreaks is usually in the cæcal region from the very onset.

I have known a patient, who had the usual pain in the right iliac region, complain of pain and even of tenderness in a corresponding point over the left iliac fossa, and now and then this left-sided (or "crossed") pain may be more marked than that over the seat of disease.

The pain may be described as "all over" the abdomen, and may go back to the loins. It is described as "burning," "tearing," or "aching." It is very acute, and has paroxysms of greater intensity. It is not like the pain of colic, although it may be spoken of as "cramp-like." It is not of a kind to bend the patient double, nor is it relieved, except at the very beginning, by pressure. Some patients compare it to an intense attack of flatulence, and they have a notion that if they could but pass wind the pain would be better.

The pain, if severe, may produce some degree of collapse. Even in mild examples the patient feels faint, a clammy sweat breaks out over his forehead, his hands and feet become cold, and he is soon quite prostrated.

Vomiting may be among the very earliest symptoms; indeed the patient may be sick almost before the pain has made itself felt. This is seen not infrequently in children. Vomiting in perityphlitis is a symptom liable to great variation; it may be entirely absent, it may occur repeatedly, or there may be only an initial vomit. It is very rarely, indeed, a severe or prominent symptom, nor is the vomiting copious or distressing. Nausea is common enough, and a complaint of "feeling sick" is usual. Vomiting may follow the use of morphia, the administration of an aperient, or the indiscreet giving of food. I have never known the vomited matter become offensive or semi-fæculent, except in severe cases which are exhibiting septicæmic symptoms, and in which there is an extensive and probably fatal peritonitis. When the bowels are loose, vomiting is usually absent.

Constipation, more or less complete, is the rule in perityphlitis. Now and then a motion may be passed at the very beginning of the attack, the initial pain being attended with an urgent feeling that the bowels must act. This sense of tenesmus is not always attended by an action of the bowels.

Sometimes the attack is marked throughout by diarrhœa, or loose motions may be passed now and then, constipation following upon such action of the bowels. Among the 190 cases dealt with by Dr. Hawkins there was diarrhœa throughout in six instances, and in eight cases out of

the 190 loose motions were passed. Thus in 7·3 per cent of the examples the bowels were not constipated. In the cases marked by constipation the bowels will often respond to an enema, or to an aperient if it be given. Speaking generally, the constipation is by no means so marked nor so obstinate as is the case in more general peritonitis.

Much tenesmus is quite rare in perityphlitis. In young patients, with acute symptoms, it may be met with as an uncommon symptom. The very worst case of tenesmus I have ever seen—worse even than in acute intussusception—was in a lad who, when the abdomen was opened by operation, was found to have a wholly gangrenous appendix.

Fever is a constant and prominent symptom. — It begins as soon as the initial collapse, if any, has passed off, and is usually of high grade. It may run up to 103° or 104°. Most often it reaches its highest point at the beginning of the case, that is, on the evening of the first or second day, and then gradually declines. This is the usual character of the chart in perityphlitis. The temperature, after subsiding a little, may rise again towards the middle period or the end of the case. These reappearances of the fever sometimes appear to be due to too early movement, or to neglect of the bowels, or to an improper diet. In other instances they cannot be explained. There is very little relation to be observed between the temperature and the local manifestations in the iliac fossa; the swelling may diminish while the fever is high or increasing; and, on the other hand, an augmentation of the swelling may be attended by no upward movement in the temperature.

The temperature may keep up for nine or ten or twelve days, and the surgeon be very apprehensive of pus. It may then subside without any evident suppuration, or any local explanation of the improvement.

The fever may continue for three or even four weeks, and be associated with no more than tenderness over the cæcal region. It then disappears as mysteriously as it was maintained. In some examples, in which the temperature has kept high, and is, let us say, at 102° on the ninth or tenth day, and then suddenly drops to normal, it is probable that an abscess existed which, on the occasion of the fall in the temperature, discharged itself into the bowel unobserved. I have found a very little pus, buried in a casing of thick and rigid adhesions and indurated tissue, to keep up the temperature for many weeks.

As a rule the temperature declines gradually, but now and then it drops quite suddenly. In the majority of the cases the temperature subsides in from six to ten days.

The range of temperature observed in the cases attended with loose or regular action of the bowels would appear to be lower.

Condition of the abdomen.—According to the extent of the peritonitis the abdominal wall becomes rigid, and the skin over it hyperæsthetic. Much tenderness is complained of over the right iliac fossa.

On attempting to examine the patient in that region he complains of great sensitiveness of the surface, and the abdominal muscles over the cæcal region are found to be contracted. At the outset of an acute case

the rigidity of the abdomen may be well marked. In any case, however, it passes off in a day or so, and the abdomen tends to become a little distended all over. This meteorism begins in the right iliac region. A disposition to contraction in the muscles of the right side on examination continues for some time.

The tenderness on the right iliac fossa persists, and indeed is present during the whole progress of the case. It is still evident, as a rule, even when all the other symptoms have passed away, and when the patient considers himself to be well over the attack. Some tenderness on deep pressure may indeed persist for weeks after the fever and all other symptoms have subsided.

When the symptoms are acute the tenderness is somewhat widely diffused over the cæcal region, but as they subside the tenderness becomes more localised. It will then be generally found located a little to the outer side of the linea semilunaris, as it crosses a line drawn from the anterior superior iliac spine to the umbilicus.

Tenderness about this spot can often be elicited on deep pressure for weeks after an attack has quite subsided, and this symptom is a valuable one in deciding the nature of a past attack. It is not infallible as indicating disease in the appendix. I have known it to be present when the inflammatory trouble was in the ovary and not in the appendix. It is desirable in all cases to remember that the tender spot does not of necessity correspond to the exact site of the appendix. When a patient is being examined for tenderness in this region, after all the essential symptoms have passed away, care should be taken to note how deep pressure is borne over a corresponding spot on the opposite or sound side; since heavy and localised pressure over this particular region is not unattended with discomfort in persons who are quite healthy.

During the attack it is needless to say that the tenderness will vary both in degree and in extent. Its intensity is not, however, a sure indication of the degree and extent of the inflammation in the iliac fossa. There may be extreme tenderness in a case which ends speedily in resolution. On the other hand, I have discovered a large collection of pus in a case in which local tenderness had never been a conspicuous feature. I have seen two cases of septic peritonitis, due to trouble in the appendix, run their entire course with a flaccid belly wall and with scarcely any tenderness. Both patients were men over fifty, no morphia had been administered, and in both cases death took place from toxæmia within seven days. One man spent some hours in applying massage to his own abdomen, and the other drew frequent attention to the absence of tenderness by slapping the abdominal wall.

As the tenderness passes off and the abdominal wall becomes less rigid, an area of dulness can be detected in the right iliac fossa, and with it is associated a more or less definite *tumour*. Both the dulness and the swelling vary greatly in extent. The dulness has its base about the anterior superior iliac spine, and this base extends inwards along about the outer half of Poupart's ligament. From this imaginary base the

dulness may extend a few inches towards the median line, or be fo reaching upwards in the region of the ascending colon. It may ass a triangular outline with its apex at or about the umbilicus: on other hand, it may reach the median line from the symphysis pubis to navel, and form a very extensive area of dulness occupying the gre part of the right half of the anterior abdominal area below the level the umbilicus. Again, it may extend round into the loin. The dul need not be absolute, nor is it indeed, as a rule, absolute in all pa especially when the area marked out is extensive. The dulness is m evident and most complete near the base above referred to. It becom less pronounced as the outskirts of the area are reached. There often patches in the defined area where percussion elicits a more or tympanitic note, and in some large part of the district the dulness be only relative. As the rest of the abdomen is usually quite t panitic, the affected area can be defined with little difficulty.

The dulness is caused in the main by effusion, by effusion bounded adherent coils of intestine and usually adherent omentum, and poss also by fæcal matter in the cæcum. It will be understood, therefo why parts of the dull area may present a resonant or relatively reso note on percussion.

The tumour is to some extent coextensive with the area of rea comparative dulness. Its base coincides with the base of the dull above given. Its lower and outer boundaries, therefore, seem to m into the iliac bone and into Poupart's ligament. Its upper and i parts are more or less distinct.

Sometimes the swelling forms a definite ball, at other time boundaries are very indistinct. It may, indeed, be no more than a of resistance associated with some degree of dulness on percussion.

The mass feels hard; and as the acute symptoms subside the swe becomes more and more definite, and then may appear as a well-ma round or oval swelling above and often parallel to the outer ha Poupart's ligament.

The swelling, as I have just said, is made up—when at its heig of inflammatory effusion limited by adhesions, of adherent coils of inte whose walls are probably œdematous, of a cæcum which very likely tains a quantity of fæcal matter, of adherent and greatly thickened o tum, and of a greatly swollen appendix surrounded by much effusion lymph.

The swelling, therefore, may be wholly dull on percussion, and dulness will always be most marked towards the base; or it may be s what resonant owing to the distension of the adherent coils of inte by gas; or even, for the same reason, quite resonant. For like rea it is often resonant in one part and dull in another, and the percu sounds may vary in the same case from time to time.

As the case advances towards recovery the effusion is absorbed lightly adherent intestines are freed, the omentum shrinks, the ca has emptied itself, and nothing remains but the diseased appendix

the more or less considerable mass of inflammatory material with which it has surrounded itself. Thus it is that the tumour becomes more and more definite as the case progresses towards recovery.

The magnitude of the swelling does not vary with the magnitude of the case. Some of the most fatal cases are attended with but little evident swelling.

The very largest tumour—one which has reached the umbilicus on the one hand, and extended to the left of the median line on the other—often subsides without producing a drop of pus. And again, an abscess may appear in the iliac fossa without any previous swelling having been noticed. There may, indeed, be no swelling at all, as is seen especially in instances in which the cæcum and appendix are lodged in the pelvis.

When the cæcum is undescended—that is, is situated beneath the liver—the swelling is in the subhepatic region.

Often in children the swelling—when of the usual form—can be felt by the finger introduced into the rectum, and in adults it may be so felt if there be an extension into the pelvis. The iliac swelling of the ordinary type cannot, however, be felt per rectum in an adult.

Occasionally, especially when aperients have been administered, peristaltic movements may be perceived in or about the tumour.

General constitutional condition.—The patient becomes more or less prostrated by the pain, by the fever, and possibly by the slight degree of septic poisoning which may be assumed to exist. The eyes become a little sunken and the face pinched. While the manifestations are acute the countenance may be that of a patient with general peritonitis. As a rule, however, the patient's aspect is not characteristic; it can only be said that he looks ill. Headache is often complained of. All appetite is lost. The thirst is not so intense as in general peritonitis. The tongue is furred, but rarely becomes dry. A most unpleasant taste in the mouth often distresses the patient. The pulse is the pulse of fever; it is seldom affected in tension, and only in quite acute cases does it become thready. The urine is that of fever, and is said often to contain an excess of indican.

The patient usually lies flat upon the back, but is often very restless. The knees may be drawn up and the hands may be placed above the head, but this attitude is never so common as it is in general peritonitis. It is not infrequent, however, to find that the patient keeps the right thigh drawn up or wishes to have it supported by a pillow.

Occasional symptoms.—The pain at the beginning may be distinctly referred to the loin, and for the moment may be mistaken for renal colic.

The pain not infrequently radiates from the right iliac fossa. The most common direction assumed is down the right thigh upon its inner side; next in frequency is a pain in the testicle, or in the external genitals along the penis or in the perineum. Now and then the radiating pain is referred to the bladder, and micturition is frequent; or, on the other hand, there may be retention of urine; this latter symptom is, I think, more common in women than in men.

There may be much hyperæsthesia of the skin over the thigh just below Poupart's ligament, or over the back of the the renal region. There may be much stiffness of the rigl limb being drawn up. In some such cases attempts to st thigh cause exquisite pain. See section on diagnosis between and hip disease (p. 925). Distressing pain in the lower par may be complained of. The attacks may be coincident with t period, and may be associated in the patient's mind with s trouble, and undoubted ovarian pain may exist. I remember t with relapsing perityphlitis complained of intense pain at tl period, such pain having been unobserved before the perityphlit in this instance I found a diseased appendix adherent to the o adhesion may exist in other cases in which there is an a ovarian symptoms; in one case I found—quite distinct from appendix—an ovary containing pus; this case had been consid

Tenesmus is quite a rare symptom. Now and then peri ments may be perceived in the abdomen, but in the great cases all is apparently still within.

Course.—The pain remains severe, and may even show s during the first two to four days; it then becomes less and The tenderness, however, remains unchanged. The fever six to nine days, and all the acute symptoms are over in ten days. In the 190 hospital cases reviewed by Dr. Hawkins ar to the present category, the average time during which the invalided or unable to work was twenty-five days. An elongat tender on deep pressure, can usually be felt in the right ilia long time after the symptoms have all vanished. The 190 recovered. In three an incision was made, and in one c appendix was removed.

II. An ordinary attack ending in suppuration.—These c the earlier days of the attack, follow closely the clinical ma just described. The onset of the attack and its principal sy exactly the same. A rigor is no more common in the cases w suppuration than in those which end in resolution. In both an initial rigor is very rare. Indeed, there is absolutely n the initial symptoms of the attack to enable the surgeon to advent of suppuration. The attack may begin with alarmi and end in ready resolution; on the other hand, the early syn be very little marked and yet suppuration occur.

I have seen a case, which at the beginning was considere

eighth day the pains became worse, and the patient took to his bed. On the ninth day a large perityphlitic abscess was evacuated.

The evidences upon which a suspicion of suppuration may be based are the following:—The general symptoms persist, and the local phenomena—as represented by swelling, dulness, induration, and tenderness—increase. The right thigh is very prone to be drawn up, and pain in the thigh or in the testicle or external genitals is more common than in the non-suppurative cases. Sometimes the pain in the thigh is very severe, and the patients cannot bear the limb to be moved or even touched. The temperature keeps high. It may fall to normal about the seventh day and then rise again—a very significant symptom. Or the tenth to the fifteenth day of the disease may be reached and the temperature still show no decline since the commencement of the attack. The patient now begins to look severely ill. The tongue, which has been much coated, becomes dry and brown. The prostration increases. When pus forms the iliac swelling is usually large, and forms rapidly. Fluctuation can only be detected when the case is well advanced, and when the tenderness and the general swelling have much diminished. Œdema of the integuments and redness of the skin are very rarely present until the other signs of abscess are quite prominent and undoubted. Furthermore, it is to be noted that a tendency to looseness of the bowels is more common in the suppurative than in the non-suppurative cases of perityphlitis.

As a comment upon the above-described evidences of suppuration the following points must be mentioned:—In the first place, an abscess may exist as early as the third day. Pus has been discovered at the end of forty-eight hours. Puckham mentions a case which ended fatally on the fifth day; an abscess was found at the autopsy which contained three pints. In the second place, pus may be present without fever, although this combination of symptoms is very uncommon. In the third place, the temperature may keep high for three or more weeks, and yet no evidences of pus be forthcoming. It is possible that in some of these cases which finally end well an abscess had burst quietly into the bowel and healed up. Fourthly, the swelling may be extensive, hard, and tender, and yet vanish completely away without the production of a drop of matter. Finally, an abscess may be present in cases in which the swelling is very insignificant. In such instances the pus may be diffused between coils of adherent and possibly distended intestine, or may be lost to view in the pelvis. If the abscess contain gas, it may exhibit a resonant note on percussion over some part or the whole of its surface. As an occasional symptom may be mentioned œdema of the right lower limb. This would appear to be due to compression of the iliac veins by the inflammatory exudation, or occasionally it may be due to actual thrombosis of that vein. Phlebitis in the lower limbs, with or without suppuration, is not uncommon in perityphlitis, and it is curious that it is more common in the left limb.

In children, and less often in adults, the swelling caused by the abscess may be felt on digital examination of the rectum.

Course.—The progress of the case is that of a deep-seated abscess. In the thirty-eight hospital cases analysed by Dr. Hawkins the following results were noted:—In five examples the abscess burst into the bowel between the thirteenth and the eighteenth days. All these cases did well. In ten cases death ensued (in four instances from intestinal obstruction due to adhesions, in one from abscess of the liver, in one after miscarriage, and in the remaining four after operation). In twenty-three cases complete recovery followed the evacuation of the abscess by operation. In one case the abscess was not opened until the seventh week.

As already stated, the abscess usually presents in the iliac region. It may, however, burst in the directions indicated in the account already given of the abscess on page 895. I have known it present in the sac of a scrotal hernia and in the inguinal canal. It has discharged through the sac of a femoral hernia. The pus may extend up along or behind the ascending colon, and may present as a lumbar abscess, and be opened in the loin. The largest perityphlitic abscess I ever evacuated was in this situation. In many instances these abscesses are actually perinephritic. The pus may extend farther up and reach the subhepatic region, and be opened or spontaneously discharge in that situation. It may go yet farther, and piercing the diaphragm may enter the pleura, or the lung. In many instances recovery has followed the natural or artificial evacuation of the abscess in this situation. The pus may make its way into the pelvis, and produce the phenomena of a pelvic abscess. The abscess may extend beneath Poupart's ligament into the thigh, and may imitate a psoas abscess, or even the abscess of hip disease. When there is stiffness of the hip-joint, and when the thigh is kept flexed, this resemblance is the more striking.

After the abscess has been well opened and drained a speedy closure of the suppurating cavity is the rule. The healing is generally complete within three to four weeks. Now and then the discharge becomes less and less, and the wound heals, and the patient is considered to be sound; but after an interval, which may vary from a few days to several weeks or even months, the abscess makes its appearance again. These relapses —if occurring at an early period—are due to the too speedy closing of the skin incision and to the resulting accumulation of matter. If the abscess reappear after an interval of weeks or months it must rank as a residual abscess, that is, as a suppurative focus in or about the residues or remains of an old abscess.

The abscess may relapse repeatedly, as the following case will show:—A lad of fifteen had a severe attack of perityphlitis in November 1892. A large abscess formed, which was incised and soon healed. In December 1892, after a period of sound health, there was a burst of pus through the well-healed scar. The opening soon healed, and the patient appeared sound. The abscess relapsed in February, April, and September 1893, after which it closed for good. Between each relapse the wound was sound and the boy well.

The perityphlitic abscess so often contains pockets and diverticles that conditions very favourable to imperfect draining and healing, and consequently to relapse, are provided by these collections. The abscess may heal several times and again relapse. In such instances it is usually necessary to secure more efficient drainage by making counter-openings, and by opening up neglected pockets. The abscess cavity will then often heal, if it be scraped out and dressed from the bottom.

Several times, in dealing with these troublesome and "relapsing" abscesses, I have come across a loose concretion when exploring the sinus, and after the removal of the concretion the cavity has at once healed and has remained sound.

Some of these cases of relapsing abscess are extremely obstinate. I have known them to continue for years, now better now worse, now healing now breaking down; and to resist every surgical measure applied to them. Fortunately such cases are not common when the large number of perityphlitic abscesses are taken into consideration.

After a perityphlitic abscess has been evacuated it is desirable to be cautious in the prognosis. It is safe to say that the majority do well, and that relapses and troubles in healing are uncommon in children. It is rather in adults that difficulties are met with.

A fæcal fistula may result from a perityphlitic abscess (page 928).

The stiffness of the thigh to which allusion has been made may persist for some time after the abscess has been successfully treated.

The œdema of the right limb also may persist for a considerable time. I have known it present for months after the abscess had entirely healed. I believe, however, that the stiffness and the œdema invariably pass off in the course of time.

For reasons which have been already explained, the patient who has had a perityphlitic abscess may be regarded as not liable to another attack of the original disease. The abscess usually removes the cause of the perityphlitis and effects a cure. I have only met with two cases in which definite attacks of perityphlitis returned in patients in whom abscess had occurred. The abscess itself, however, may give trouble, and those patients who are the subjects of "fresh abscesses" weeks and months after the original incision has closed, must be regarded not so much as the subjects of relapsing perityphlitis as the victims of an intricate abscess which has not undergone complete obliteration.

III. An attack of the mildest type.—Such an attack consists of more or less sudden pain in the abdomen, some tenderness in the right iliac fossa, a little rise of temperature, and possibly some vomiting. The patient is laid up for a few hours, or perhaps for one or even two days. He is then well, and is only conscious of some tenderness and stiffness in the right iliac fossa. Such attacks are often described by the subjects of relapsing perityphlitis. The symptoms are not severe, but for an hour or so at the onset the patient may feel very ill, and there may even be a slight degree of collapse.

The attack is often preceded by malaise and a complaint of flatulence,

and even of colic. Constipation is usual, and if seen during the attack or immediately after it the tongue is found to be furred.

One patient—a man of thirty—who consulted me had had sixteen attacks of the type above described. They lasted from one to three days at a time, and the symptoms were so mild that he was never laid up for more than an hour or so. There was no vomiting. In another case—that of a man of twenty-four—twelve attacks occurred in two years. In each attack there were the symptoms above detailed, and in each attack there was vomiting. As soon as he had been sick the trouble began to pass off; on each occasion there was a rise of temperature. The patient was never laid up for more than a few hours.

As illustrations of attacks of a somewhat severer grade the following may be selected :—

A man, aged twenty-two, when twelve years of age, had a severe attack of perityphlitis, which laid him up for three months. No abscess formed. Ever since, that is, for a period of ten years, he had had from six to eight attacks every year.

His attacks begin with uneasiness in the belly, then a very severe pain is experienced which locates itself in the right iliac fossa. This region of the abdomen becomes hard and tender, and the thigh is drawn up. There is nausea invariably, but no vomiting. The temperature rises to about 101°; it has registered 103°. The patient has an injection of morphia and is soon easy. He lies in bed for from thirty-six to forty-eight hours, and then goes about his work, but for a day or so he has little appetite, and is indisposed to walk. The attacks are attended by constipation. Besides these major attacks, every three weeks or so he has minor attacks marked by some little pain in the right side, loss of appetite, nausea, and disinclination to move about. The attacks never lay him up and never require morphia.

A lady, aged thirty-five, who had always been subject to looseness of the bowels and some dyspepsia, had eighteen "attacks" in five years. After a restless night she would experience in the morning a sense of malaise. She would eat an indifferent breakfast, and after the meal would feel chilled, have a loose motion, and begin to feel pain in the right iliac fossa which increased rapidly in intensity. About noon or some time in the afternoon she would have to go to bed. The temperature would vary from 99° to 100°. There would be nausea, but no vomiting. The bowels would become confined, and the right side of the abdomen very tender. The treatment consisted of poulticing, abstinence from food, and the use of opium. The patient would keep in bed from two to five days. For some days afterwards the stomach would feel too tender to permit her to wear corsets. In this case the appendix was very distinctly palpable.

By some writers these short—although at the time severe—attacks, which last twelve, twenty-four, or thirty-six hours, are described as "appendicular colic." Appendicular colic was first described by Melier, who considered the pain to be due to distension of the appendix.

Talamon, writing in 1890, considers that the sudden and sharp pain in the cæcal region, attended by local tenderness and possibly by vomiting, is due to calculus in the appendix, and is to be compared to gall-stone colic or to the colic of renal stone. Other writers have considered the pain to be due to an accumulation of mucus in the appendix.

All, however, are of opinion that the suffering is due to a spasmodic contraction of the organ, whether it be attempting to expel a calculus or to rid itself of an accumulation of mucus. They regard it as a real colic comparable to intestinal colic. The cessation of the pain is supposed to coincide with the emptying of the appendix or the passage of the stone into the cæcum.

A study of the pathology of the appendix makes it evident that appendicular colic has an extremely doubtful existence. The appendix itself is not sensitive; that which feels the pain is the peritoneum over it. The muscular apparatus of the appendix is feeble at its very best, and when the appendix has been long diseased it may be said to have no existence. The notion of an appendix emptying itself of an accumulation of mucus or muco-pus by such violent muscular contractions as would cause intense colicky pain is wholly ridiculous. The appendixes which contain muco-pus have little trace of efficient muscular tissue left, that tissue being scanty enough in the sound organ. The idea advanced by some pathologists that the calculus enters the appendix from the cæcum is based upon a pathological error; the calculus is always formed in the appendix. Those who have examined the wall of an appendix in which a calculus has lodged could never be induced to believe that the infiltrated wall with its doubtful trace of efficient muscle could contract upon the calculus with so violent a grip as to expel it into the cæcum, and at the same time give rise to a pain sufficiently severe to produce some degree of collapse.

In short, there is no evidence, either clinical or pathological, to support the existence of "appendicular colic." The term is as ill-judged as it is erroneous.

IV. An attack of the most intense and acute type.—In these attacks the appendix is found to be perforated; or to have given way entirely at a line of stricture; or to have been burst, as it were, by a calculus; or to have become more or less entirely gangrenous from causes which have been already detailed (page 887).

It may be found quite detached.

Peritonitis is discovered in these cases to have become more or less general, and to be of an ill-conditioned kind.

The onset may be—and indeed usually is—of the degree described in dealing with the ordinary attack. It may be very severe, producing intense symptoms and marked collapse. As a rule, however, as I have said, the severity of an attack cannot be judged by its mode of onset. Attacks which begin with the most alarming symptoms may end mildly, and on the other hand, a case fatal in a few days may begin in a manner which excites no alarm. The symptoms, however, in the present

case soon become marked by their severity. The temperature may ra to 102°, to 103°, or higher. In the cases which present markedly sep phenomena almost from the first the temperature may not rise abo normal, and may soon become subnormal. There is often considerab vomiting with increasing collapse. Finally, there are all the evidences general peritonitis and the general symptoms of poisoning which ma the fatal examples of this disease. Death may take place very rapidly Earle mentions a case in which death ensued in thirty-six hours.

I saw a gentleman aged twenty-eight who, after three weeks "dyspepsia," during which time he was actively engaged in some a sorbing out-door work, took a long railway journey on a certain Saturda He reached his destination on Sunday evening, ate a good dinner, a sat up smoking until midnight. At 2 A.M. on Monday morning—th is, two hours after retiring to rest—he was seized with definite sympto of acute perityphlitis. At 2 P.M. on the same day when I saw him was cold, pulseless, and dying. At 6 P.M. he was dead. Here de followed in sixteen hours. At the autopsy a diseased appendix was fou to have given way.

Cases such as these must rank with examples of acute perforati peritonitis. In other instances the symptoms for several days are notably severe nor especially alarming, but then the phenomena of ac perforative peritonitis suddenly appear and rapidly advance to a fa termination. In such cases no doubt a gross perforation or an extens and rapid gangrene of the appendix lights up the severer train of sy toms. I have known such a sudden accession of fatal symptoms foll the use of violent purgatives in a case which was apparently doing we

A very large proportion of these fatal and severe cases are first atta Those who have had many attacks seem to be better able to bear a w spread peritonitis, or are perhaps locally protected against it to s extent by old and dense adhesions around the diseased appendix. of the thirty-six cases collected by Dr. Hawkins no less than twe seven patients died, and in all of them the phenomena at the close of case were those of diffused peritonitis. Four of them died on the f day, three on the sixth, and seven on the seventh day; one succum on the eighth day, three on the ninth, and the remaining nine patie died at periods between the tenth and the nineteenth days. It is evid that in the cases in which death was postponed until the fifteenth nineteenth day the peritonitis must have remained for a long time lo and only become general towards the end. In the examples of de within a week of the onset of the trouble the peritonitis was very p ably general from the first.

It will be seen, therefore, that these very acute and severe form perityphlitis may be fitly described as examples of perityphlitis atten by general peritonitis, that peritonitis being, in many of the instances doubt, perforative.

V. Certain peculiar forms of perityphlitis.—Under this head may be considered—A. The masked cases; B. The chronic cases; C.

cæcal form of perityphlitis; D. The tuberculous form; E. Perityphilitis due to actinomycosis; F. Relapsing perityphlitis.

A. *Masked cases of perityphlitis.*—In these cases, which are fortunately uncommon, practically all the ordinary phenomena of perityphlitis are absent. One of the most extreme examples of this insidious form which I have met with was in the person of a middle-aged gentleman, who after being "a little out of sorts" was seized with pain in the hepatic region, attended by a rigor and subsequently by a rise of temperature. He very reluctantly kept to the house. In due course the rigors were repeated, the fever became very high, jaundice supervened, and the patient became exceedingly ill. It became evident that he was suffering from pylephlebitis. No mischief of any kind could be detected in any part of the abdomen except about the liver. The right iliac fossa had been repeatedly examined. In fourteen days he died. The liver was found riddled with minute abscesses. The appendix was utterly disorganised and filled with pus; it had evidently been the seat of long-standing disease. I can definitely assert that the patient never had a single symptom proper to perityphlitis, and yet this was the primary disease which led to his death.

In other less extreme cases there are no phenomena which are in any way characteristic or even suggestive. There are no sudden onset, no definite pain in the right iliac region, no fever, no very noteworthy tenderness, and no characteristic swelling. These patients complain of feeling ill; they are listless and out of sorts; they still attend to their business, but with effort. They complain of loss of appetite and constipation and very probably of vomiting. The tongue is furred, the belly is a little swollen, feels full, and is frequently the seat of indefinite colicky pains. There is no fever, or if there be it is of very slight degree. Possibly there is tenderness on deep pressure in the right iliac fossa. A swelling may be noted and ascribed to a loaded cæcum, the bowels being much confined. These cases go on until one day an abscess is discovered, and then the diagnosis is made.

Some of the cases met with in sickly children are very insidious and very misleading.

B. *The chronic cases.*—The chronic cases now to be considered must be distinguished from relapsing perityphlitis. In relapsing perityphlitis acute or subacute attacks follow each other in a series. They come on at indefinite intervals, and lay the patient up for varying periods. When, however, the attack passes off the patient makes a sound recovery, and between the attacks he is well. He may remain in health apparently perfect for months or even years, should the intervals between the individual attacks be so long.

In chronic perityphlitis there are attacks or relapses, but the patient is never well between these manifestations of more acute trouble. The affection appears as a chronic disease with somewhat indefinite exacerbations. It is best illustrated by a few examples. A metal polisher, aged nineteen, came to the London Hospital in April 1895. He had had his first

definite attack of perityphlitis in October 1894; it came on afte severe bout of sea-sickness. He vomited and his bowels were constipat but he was only confined to bed for two or three days. There we marked pain and tenderness in the right iliac region. He returned to h work on the fourth day, but never felt well. The pain in the lower pa of the abdomen persisted and he was always in discomfort. He got abo with effort. In November he had a much sharper attack, attended agai by vomiting, and associated with troublesome constipation. There we fever and much local tenderness. The least exertion brought back t pain in the cæcal region, with a sense of nausea and faintness. Fro November until the following April he had been an invalid, and had no been able to do one hour's work. For five weeks, indeed, he kept in be When he attempted to get about again the pain came on in the cæ region, and he felt sick or actually vomited. The bowels were mo obstinate, colicky pains were common, the belly was never free fro discomfort, the tongue was coated, the appetite was poor, dyspepsia w complained of, and some fever at night was common. The patient w least uncomfortable when lying down, and for nearly five months w hardly able to leave his bed or sofa. The right thigh was kept flexed the hip, and the right iliac fossa was always tender. The patient becam wasted and cachectic-looking, and very "nervous." I removed th appendix in May, seven months after the first attack. It was buried a remarkable mass of the densest adhesions, and was represented by tract in the centre of this mass filled with pus, blood, and mucus.

In another case a married woman, aged thirty-one, had been confin to her bedroom for over twelve months, and had in the meanwhile be delivered of a child. She began her troubles with a definite attack of pe typhlitis, and then passed into the chronic condition above described. Soc slight degree of fever was nearly always to be noted at night. She w not ill-nourished, and while she kept at absolute rest in bed she had n much trouble, but the least movement brought on pain in the right ili fossa, with nausea and malaise, and a more definite rise of temperatu Over twenty times within the twelve months the patient endeavoured move above her room, but always with the same result. Tenderness w always to be made out over the site of the appendix. I removed t diseased organ and found it adherent and full of thick pus.

A case reported by Dr. Kelynack reveals symptoms of a graver ch acter and more extensive mischief within the abdomen. A married wom aged fifty-four, was admitted into the Manchester Infirmary on August 1892. For no less than ten months the patient had been troubled attacks of vomiting which occurred almost every day. No cause for su a symptom could be suggested by the patient herself, and no history acute illness could be obtained. During the last three months the vomiti had been much worse, and she had lost weight rapidly. Two months a she first noticed that the abdomen was distinctly enlarged. On admissi she was in an extremely weak and almost collapsed condition. Vomiti was frequent. The abdomen was distended, and in greater part dull

percussion. The pain, such as it was, lay in the right hypochondrium and epigastric region. The temperature was normal. The vomiting persisted, and the patient sank and died three days after admission. The autopsy revealed an enormous perityphlitic abscess containing several quarts of foul-smelling pus. The abscess reached to the liver, occupied the pelvis, and extended far to the left of the median line. The appendix was bound down by old adhesions and was perforated; the stomach was congested.

C. *The cæcal form of perityphlitis.*—These cases cannot be quite so precisely defined clinically as might be wished; that they are rare I have already said, and as in the majority of instances they end in complete recovery actual inspection of the parts is very rare. The account which follows is based upon an investigation of the few cases in which it was possible to demonstrate by operation that the appendix was to all appearance entirely healthy. One such case was the following:—A neurotic and dyspeptic man of twenty-eight had had several mild attacks of perityphlitis. He attached a very exaggerated importance to these outbreaks, and regarded them as desperate illnesses. He was about to leave England for a distant colony, where he would be living almost beyond the confines of civilisation, and he was exceedingly anxious to have his appendix removed before he left. The precise physical condition did not render such an operation necessary, but the mental attitude of the patient was such that it was rendered at least politic and desirable. The abdomen was opened, but nothing abnormal was discovered except a few loose adhesions about the cæcum. The appendix appeared perfectly normal, but I considered it desirable to remove it; it proved on minute examination to be healthy. The patient made a good recovery, and has never had another attack of perityphlitis.

The subjects of this variety of perityphlitis are usually dyspeptic and the subjects of constipation. They often have imperfect teeth, and bolt their food. They often pass scybala. They are troubled with flatulence. The attacks may come on after a definite error in diet or an unusual neglect of the bowels. Not a few of the patients appear to have a chronic form of mild colitis and pass much mucus.

The attack comes on gradually rather than violently and abruptly; the pain at the outset is not at its worst, but becomes gradually more and more severe. The symptoms are loss of appetite, coated tongue, and, possibly, vomiting, with fever in slight degree. The tenderness in the right iliac fossa is somewhat diffused, and some swelling is to be discovered, as a rule, almost from the first. This mass can often be made out to be a fæcal collection in the cæcum. The stools which are passed after the attack are most offensive, and contain scybala. The breath is offensive. The attack, which is of a mild character, passes rapidly away, and, save for some soreness in the right iliac region, in a few days the patient is well.

Now and then the subjects of many of these slight attacks affirm that indulgence in certain articles of food or neglect of the bowels will

precipitate an attack. I am aware that such conditions are not conclusive evidence that the mischief has its origin in the cæcum, but the circumstance has been noteworthy in instances within my own experience in which the appendix was demonstrated to be free from disease.

The attack is certainly not always of this mild character, and may assume the features of an ordinary perityphlitis. One such case—in a young man aged nineteen—is detailed below. Here the symptoms of the attack were of the ordinary type. There was nothing to suggest that the cæcum was primarily at fault. An abscess formed, and on its evacuation it was made evident that the cæcum was at fault while the appendix was healthy.

On the other hand, mild attacks of the character just described as common in the cases of cæcal origin may be met with in association with definite disease limited to the appendix.

The admission, therefore, has to be made that perityphlitis of cæcal origin may be suspected, but cannot be certainly diagnosed from the clinical manifestations alone.

D. *The tuberculous form.*—The symptoms of tuberculous perityphlitis may be of the ordinary kind, and may present no especial and distinctive characters. In a certain number of instances, however, in which I have demonstrated the condition on operation the symptoms have been of an insidious and chronic character. The following case may be taken as a type of the examples now under consideration :—

A lad of nineteen, a member of a tuberculous family, was admitted into the London Hospital with the following history. He had always been thin and delicate. He had been ill for nine months. Before that time he was able to lead an active life. Nine months ago he began very gradually to be troubled with colic, and to have a more or less constant uneasiness in the abdomen. Diarrhœa became a conspicuous symptom, and some tenderness and dulness were discovered by his medical attendant in the right iliac fossa. During the nine months he was quite an invalid; sometimes better, sometimes worse. He was confined to his bed for weeks at a time, and never left the house until he came to the Hospital. He had wasted, his appetite was poor, food gave him pain, he was occasionally sick, his tongue was red and raw, and there was continual abdominal uneasiness; this, which now and then amounted to severe pain, was in the right iliac fossa, where there was always a tender area. He had diarrhœa, attended by much mucus, but now and then there were periods of constipation. There were no rigors, but always some fever, with, at last, flushings and sweating as in hectic. Indeed, he presented, on admission, the general phenomena of hectic fever. There were no signs of tuberculosis in the lungs. The abdomen was not swollen, and there was no ascites. The right iliac region was tender; in it could be felt a vague resisting mass, which was resonant in a modified degree on percussion, and which was described in my notes as "feeling like a cæcum made of leather." Near it were two round lumps in a line, each lump being about half an inch in length. They were considered to be

concretions in the appendix, but they proved to be enlarged and almost calcareous tuberculous glands. I performed abdominal section, and found a cæcum with greatly thickened and leathery walls studded with tuberculous nodules. The omentum was adherent to it. The appendix was apparently normal, but it exhibited tuberculous changes on incision. There were many tuberculous glands besides those mentioned. There was no tuberculous infection of the peritoneum. The trouble, indeed, was almost entirely confined to the cæcum.

In another case, also in a young man, the patient had experienced a very similar chronic illness, with symptoms almost identical. In addition, however, he had had several attacks of a subacute character, which were always regarded as due to mischief in the appendix. There was a large area of dulness on the right iliac fossa, with tenderness and abiding fever. On opening the abdomen I found the cæcum and appendix buried in a mass of adhesions and studded with tuberculous nodules. The infection had spread to the general peritoneal cavity, and had evidently extended from the cæcal region as from a centre. The appendix was apparently much enlarged, but the extent and density of the adhesions, and the fact that the organ was lying on the iliac vein, prevented me from removing it. There was some ascites. The patient made an unexpectedly good recovery, and when heard of, some two months after the operation, was reported as "a great deal better."

E. *Perityphlitis due to actinomycosis.*—The best-recorded case of this rare condition is that detailed by Dr. Ransom, and already alluded to (page 890). The patient was a man of fifty. His illness began on 12th October 1888 with severe but somewhat indefinite abdominal pain. He continued to get about until 21st October, when he was seized suddenly with defined pain in the right iliac fossa. On examination, one hour after this acute onset, Dr. Ransom found in the cæcal region a firm, uneven, solid mass, tender on pressure. The patient gradually improved, and was able to get up on 18th November. On the evening of this day the temperature rose a little. The swelling increased, and involved the whole iliac fossa, extending upwards to above the iliac crest. There was great local tenderness. The temperature varied greatly, rising sometimes to 102·8° F. The pulse ranged from 84 to 90. On 5th December he was again suddenly seized with severe pain in the cæcal region, which also extended down the right leg, and it soon became evident that there was thrombosis of the external iliac vein. On 9th December pus was suspected, but aspiration revealed nothing. The swelling increased, and the right loin, buttock, and the upper part of the right thigh all became much swollen. On 14th February an abscess pointed and burst. About an ounce of thick, dirty pus with a fæcal odour was discharged. The patient was relieved. Fæcal matter was discharged from the wound. On 20th March more pus was liberated from the wound. The patient did not improve. He became gradually worse, and died on 21st June.

In one case, which I had an opportunity of seeing early, the patient, a middle-aged man, had the same equivocal symptoms at the commencement.

He did not begin with a definite sudden attack. There was a considerable swelling in the right iliac fossa, and as the phenomena of inflammation were not of a degree corresponding to the size of this swelling, there was a strong suggestion that the mass was a growth. Any surgeon would have hesitated between the diagnosis of a growth about the cæcum and very deep-seated pus surrounded by a considerable mass of adhesions. The patient had been losing energy and strength for some time before the swelling in the right iliac fossa appeared [*vide* art. "Actinomycosis," vol. ii. p. 81].

The local swelling would always seem to be a very early symptom, and to be out of proportion to the subjective symptoms.

F. *Relapsing perityphlitis.*—This name is conveniently applied to cases in which the patient has many separate attacks of perityphlitis. About the attacks themselves there is nothing in any way noteworthy. The determining clinical feature is that the attacks are repeated. Perityphlitis constituted by a single attack is more common than in the relapsing form. Dr. Hawkins, in dealing with the histories of 250 cases, found that in 23·6 per cent there had been more than one attack. Fitz calculates that recurrence is met with in about 44 per cent of the cases, and I am under the impression that this higher percentage is the more nearly accurate. Age has no concern in favouring recurrences, and there is nothing about the first attack which makes it possible to foretell whether there will be any relapse.

In relapsing perityphlitis the patient is practically well between the attacks. In this respect the condition differs from that described as chronic perityphlitis, in which the patient is never free from more or less discomfort, local or general (p. 913).

The individual attacks may be of any degree; they may be very severe, they may be of the type described in the above account as "ordinary," or they may be of the mildest possible character. Any one attack may lay the patient up for months on the one hand, or be so trivial on the other as to merely cause him to rest for a day. After many attacks one may occur which ends in suppuration. As already stated, when once an abscess has formed there is seldom a genuine relapse. The abscess may give trouble, may seem to heal, may break out again, and heal again once more to burst forth; but these relapses are not definite attacks of perityphlitis, they merit the name rather of relapsing abscesses.

The number of the attacks, the intervals between them, and their relative gravity vary in an extraordinary manner. A patient may have fifty attacks in five years. There may be fifteen attacks, or even more, in twelve months. Such frequent attacks are of course trifling in severity. On the other hand, ten years may pass by with but three attacks, of which one may be severe and two trifling. The infinite variations met with in relapsing perityphlitis may be illustrated by the following case:—The patient was a middle-aged man. His first attack was in 1874, and was very severe. There then followed a series of

trifling attacks that laid him up for one to three days only. In 1877 he was the subject of a very grave attack, and was laid up for four months. In 1878-79-80-81 and 1882 he had numerous trifling attacks, which were annoying rather by their frequency than by the distress caused by the individual outbreaks. The patient was then entirely free from his old trouble for five years. In 1888 he began again to have slight attacks: they occurred every few months or weeks, and on each occasion he was confined to his bed for from one to three days. In October 1893 he had another severe attack, and was confined to bed for four weeks. In November I removed the appendix. This patient had therefore been troubled with relapsing perityphlitis for nineteen years, during which time he had had three severe attacks and countless trifling ones.

At the operation, on account of the considerable amount of thickening which was to be felt in the iliac fossa, the incision was made in the median line. There were extensive adhesions, and much omentum was cut away. The tip of the appendix—greatly thickened—was adherent to the cæcum. When it was freed two perforations in the cæcum were discovered which were closed by sutures. Behind the cæcum a cavity was discovered filled with a custard-like substance. This collection extended as far down as Poupart's ligament. To the wall of this cavity the ileum was adherent. The cavity was scraped out and dusted with iodoform. The operation lasted one hour and a quarter. The patient made a sound and speedy recovery.

In other instances the patients have had three severe attacks in twelve or eighteen months, each attack being more grave than the one which preceded it.

It is common to have a relapse very soon after an attack, especially after a first attack. These examples, however, do not strictly belong to the present category; literally they represent a relapse of the original attack from which the patient had never entirely recovered, and they are often due to indiscretion in diet, or to premature moving about after confinement in bed.

Sometimes the recurrent attacks in women are coincident with the appearance of the menses.

Between the attacks the patient is usually well; but he is probably constipated and liable to dyspepsia, and deep pressure in the right iliac fossa usually elicits some tenderness; other patients have slight attacks of colic; a few complain of a weakness about the right groin, or there is actual discomfort in the right iliac fossa, noticed especially after exertion, which may prevent them from walking far. Not infrequently there is an uneasy catarrh of the bowels, and considerable quantities of mucus may be passed together with intestinal casts. Often there is evidence that the muscular movement of the cæcum is embarrassed; probably by adhesions which interfere with the normal peristalsis. In such cases the cæcum may be found distended with fæcal matter, or it may be empty and much distended with gas, or gurgling may be evident upon pressure over it. The patient is aware of some uneasiness in the cæcal district.

A condition of hypochondriasis is not uncommon in relapsing pe typhlitis, especially in men. They become absolutely absorbed in the symptoms, which they greatly exaggerate, and which they are perpetuall endeavouring to interpret. They have—or profess to have—a conscious ness of intestinal movements, which can only be described as super natural.

Very commonly in the relapsing cases the diseased appendix can b felt between the attacks as a more or less distinct, firm, fixed and tende body. When it can be readily felt it is safe to predict that the patien will have another attack; when it is not only readily felt, but is als habitually tender and the seat of discomfort, it is safe to predict tha it contains pus.

A little care must be taken to identify the appendix in these i stances. Deep pressure with the tips of the fingers in the iliac region these cases often appears to promote localised contraction of the muscl of the parietes. Such contraction may simulate a deeply-plac substance about the size and length of the little finger, and having nearly vertical position. The muscular bundles which can effect th imitation belong, I think, usually to the external oblique muscle, but m be represented by the outermost fibres of the rectus. It is well, the fore, to be suspicious of an appendix which is felt as a rounded rod a which is vertically placed. I was first made aware of this condition in case of relapsing perityphlitis in which I had diagnosed an enlarg appendix so placed. When I came to operate I found the situati I had indicated free from disease; the cæcum and the ulcerat appendix were deep in the pelvis and far beyond reach from the surfac

I have also mistaken a roll of adherent omentum for a diseas appendix, and enlarged glands for an appendix containing a concretion presenting great distension of its walls. A supposed appendix has, operation, turned out to be a little mass of hard fæces in the cæcum.

In one case of relapsing perityphlitis, which I was carefully watchi the appendix became very evident during the attack, and remai palpable for some weeks after the patient was perfectly well again. time, however, it became impossible to feel it, even on the most care examination. On one occasion I was examining the patient at a ti when she appeared to be in perfect health, and I was surprised discover the appendix, which had long vanished, again in evidence. T next day it was very much larger, but the patient was still quite w On the third day another attack of perityphlitis set in suddenly a acutely.

The condition presented by the appendix in these cases varies great and it is impossible from the symptoms to forecast the morbid appearan to be discovered on operation. In cases in which the attacks have b very severe the appendix has been removed with the greatest ease, a has exhibited comparatively little morbid change. On the other hand have found in patients who have had mild attacks an almost rot appendix surrounded by adhesions of the densest kind.

In one instance in which the attacks had been very acute I found a long appendix with a perfectly free lumen, but bent upon itself to a right angle by some few adhesions.

Again, in patients whose attacks had been so slight as to make the justifiability of any operation a matter of question, I have found the appendix almost impossible to remove.

I have removed the appendix in relapsing perityphlitis during an interval of apparent health over 150 times since I first introduced this particular operation in 1887. The condition found in these cases is tabulated as follows :—

	Per cent.
Appendix stenosed and the distal parts dilated	30
Appendix bent upon itself and the distal parts dilated and much ulcerated	15
Appendix constricted or bound down by adhesions, and the distal part dilated and much ulcerated	8
Appendix twisted upon itself and the distal part dilated	4
Appendix buried in a mass of dense adhesions, and extensively diseased or partially destroyed	16
Appendix occupied by a concretion	15
Appendix little changed, and exhibiting only the phenomena of ulceration	4
Appendix ulcerated and perforated with an abscess outside its walls	8
	100

It would appear as if, in the majority of cases, the attack is due to distension of the appendix behind a stenosed or occluded point. This distension reaches a certain maximum and an attack is the result; the tube then possibly empties itself by perforation or by the restoration of the canal, and this intermittent distension, repeating itself, explains subsequent attacks.

The dilated appendix need not, however, empty itself, for I have often found in exposing the process during a quiescent period that it is so distended as to be as hard as a stone.

DIAGNOSIS OF PERITYPHLITIS.—There are certain symptoms which, at the very outset of the trouble, are common to nearly all acute disorders within the abdomen in which a sudden and violent impression is made upon the great abdominal nerve-centres. These symptoms consist of intense and sudden pain in the abdomen, of collapse in varying degree, and of some vomiting. At the very outset such conditions as perityphlitis, renal and gall-stone colic, twisting of an ovarian pedicle, torsion of a movable kidney, general peritonitis, and intestinal obstruction have been confused one with the other. This confusion, however, is seldom long-abiding. In a little while, often in a few hours, distinguishing symptoms make their appearance and the diagnosis is established. When, however, these distinguishing symptoms are ill marked, and when imitative symptoms appear in their place, confusion is very possible.

Perityphlitis and *renal colic* have been many times confused. The pain in perityphlitis may be referred to the loin, and when the inflamed appendix is situated in front of the kidney there may be tenderness over the renal area. In perityphlitis the pain may extend to the external genitals (not usually to the testicle) and to the bladder. There may be frequent micturition. It is especially to be noted that this extension of the pain is not common quite early in the attack.

In renal colic—as compared with perityphlitis—the pain is very intense and is apt to persist without variation. It runs almost at once down to the groin, testicle, and bladder. The bladder is frequently irritable. The urine contains blood. The testicle may be retracted. There is no fever. Vomiting is not a common feature. The pain often ends suddenly. There may be previous symptoms suggestive of renal stone.

Tenderness, if present, is more marked when the examination is made over the dorsal aspect of the kidney than over its abdominal aspect.

In *gall-stone colic* the following features may be noticed which should distinguish the condition from an attack of perityphlitis :—

In gall-stone colic the pain is very sudden and a rigor is not uncommon; the vomiting is usually more severe; there are pains or "spasms" in the stomach or referred to the stomach, and pains which radiate to the scapular region or to the shoulder. There is often tenderness over the gall-bladder. There may be jaundice. The history of the case and the age of the patient may give some guidance. Much importance must attach to the fact that the right iliac fossa is clear both of tenderness and swelling. The gall-bladder, however, may find its way into the right iliac fossa. In the case of a woman aged thirty, at the London Hospital, I found in the situation of the appendix, and reaching down to Poupart's ligament, a banana-shaped swelling which was movable, which had a distinct lower end, and which appeared to be about three-fourths of an inch broad and three inches long. The patient had never had jaundice, but she had had three attacks of "gastritis." The case was sent in as an example of disease of the appendix. The tumour proved on operation to be a distended gall-bladder containing three very large stones.

Perforation of an ulcer of the stomach has been diagnosed in a case in which perforation of the appendix led to sudden and intense pain, with collapse and subsequent vomiting; the pain being seated, as is not uncommon in the early stages of perityphlitis, above the umbilicus. In one case of this kind, in which I was called upon to open the abdomen over the stomach, the patient—a woman—had had in previous years an ulcer of the stomach. The first matter vomited was said to have contained blood, and all pain was complained of above the umbilicus. When the patient was under the anæsthetic I detected a little dulness and resistance in the right iliac fossa, and an incision made over it revealed an almost gangrenous appendix perforated at its base.

Typhoid fever.—I have met with several instances in which perityphlitis had been mistaken for typhoid fever, but only one instance in which genuine typhoid fever was mistaken for perityphlitis.

It is needless to say that the phenomena of typhoid fever are liable to considerable variation, that the fever may be attended by peritonitis in the right iliac region, and that there may be perforation of the bowel in that quarter. Sir Wm. Broadbent, in his account of typhoid fever in Quain's *Dictionary of Medicine*, says that "he has known several instances in which patients have *walked* into the London Fever Hospital with perforation." The patient with typhoid fever may suffer, indeed, so little depression of strength as to keep about and follow his usual avocation until a late period of the attack. Perforation in such cases may not unreasonably be attributed to mischief in the appendix.

On the other hand, the cases of perityphlitis which may imitate typhoid fever are those in which the advent of the symptoms is not abrupt, in which the progress is insidious, and particularly in which septic symptoms are in evidence. Very often there are misleading features in the patient's history. In one case the patient had been nursing a sister with typhoid fever, and was confident that she had contracted the disease. In many examples of perityphlitis there is diarrhœa, and, if so, another element is added to the resemblance of the two complaints.

In discriminating between the two affections stress must be laid upon the following features in typhoid fever, features which are not met with in the appendix affection. The onset of the fever is slow, and there is much malaise during the period of invasion; yet this is not inevitable, as the invasion may be abrupt. The course of the fever is very characteristic when it is well marked. The tongue is seldom imitated in perityphlitis. Finally, in the infective fever are to be noted the peculiar stools, the enlarged spleen, and the rose spots. Vomiting is not common in typhoid fever, and the amount of local tenderness is very rarely extreme.

Colitis or *membranous colitis* may present some resemblance to perityphlitis, especially when the cæcum and ascending colon are conspicuously affected. The trouble manifests itself by periodical attacks alternating with moderately good health. The attacks may last a few days or a few months; they may come without reason, or may follow indiscretions in diet, constipation, fatigue, and cold. They are constituted by abdominal pain and tenderness over the affected part of the colon, vomiting of a mild degree, depression, and the passage of loose motions containing much mucus or "shreds," or "casts," or even blood. Unlike perityphlitis the onset is not sudden, the pain is not severe, and is more like that of colic; the tenderness is slight and is diffused; the temperature is seldom raised, and the motions are characteristic.

Intestinal obstruction.—In a great many instances perityphlitis has been mistaken for acute intestinal obstruction. An operation has been performed to relieve the obstruction, and a diseased appendix has been discovered. On the other hand, it is comparatively rare for actual intestinal obstruction to be mistaken for perityphlitis.

In acute intestinal obstruction—as compared with perityphlitis—the pain, as a rule, is more acute, and is apt to produce a marked degree of collapse. There is greater prostration, the abdomen becomes more rapidly distended, vomiting becomes much more copious and more persistent, the temperature is rather below normal than above it, and localised tenderness in the abdomen is usually absent.

In children acute intussusception may bear a close resemblance to perityphlitis. The pain is often confined to the right iliac fossa and in this situation a lump can easily be felt. Moreover, I have noted marked tenesmus in acute perityphlitis in children. In the differential diagnosis the following points should be noted:—In acute intussusception the pain is rather that of severe colic, and is often relieved by pressure. Fever is uncommon, and the temperature is often below normal. The tumour which may be felt in the right iliac fossa is well defined, and is above all movable; it is apt also to change its position. In intussusception vomiting is much more marked, and moreover there are in this disease the two conspicuous symptoms of blood-stained stools and continuous tenesmus. The tenderness in the right iliac fossa is not so pronounced in intussusception, and may indeed be absent; in any case it bears no relation to the size of the swelling.

Pelvic peritonitis.—The appendix may be lodged in the pelvis, and when diseased, may produce definite localised pelvic peritonitis. The circumstances of such occurrence may lead to an error in diagnosis. As an instance I may mention a case in the London Hospital, in which perityphlitis, due to disease of an appendix lodged in Douglas' pouch, supervened four weeks after a severe confinement from which the patient at the time had hardly recovered.

Tuberculous peritonitis.—This disease may present a very close resemblance to perityphlitis, especially to the chronic cases. As an instance the following example may be given:—A sickly-looking lad of thirteen was admitted into the London Hospital with this history—had pain in the abdomen, with occasional sickness and obstinate constipation. He declared that the pain came on suddenly some months previously, that there was a tender swelling in the cæcal region, and that he was sick. He went to bed and his condition soon improved, although he had never felt quite well. As soon as he attempted to get about again the inconvenience in the right side returned, and although he did not actually take to bed again he moved about with constant discomfort. He was thin, and in the right iliac fossa a considerable, ill-defined, tender lump could be made out; it was dull on percussion. The belly was a little swollen, and the seat of a general and constant uneasiness. There was slight fever. He vomited now and then, and there was most obstinate constipation.

An exploratory incision revealed a localised tuberculous peritonitis with evidences of extension over the general serous surface, and with an apparently sound appendix.

Very often in the cases in which tuberculous peritonitis begins

the right iliac fossa the appendix is diseased, and the case is a genuine one of tuberculous perityphlitis.

Cancer of the cæcum may produce perityphlitis by the production of a perforation, and upon this an abscess usually follows. The onset, however, is seldom acute; the swelling and some intestinal disturbance precede the acuter phenomena. There is seldom much fever. The patient is wasting and has been out of sorts for some time.

Hip disease.—It is remarkable that perityphlitis in children may—for a time at least—resemble hip disease. I have seen many such cases. Yet I have never seen or heard of a single instance in which genuine hip disease had given rise to a suspicion of perityphlitis. In the majority of the cases of perityphlitis which have given rise to a suspicion of hip disease there has been an abscess, but this is not invariable.

The cases in which a resemblance to hip disease is marked are the following:—The patient, usually a child, is seized with pain about the right hip bone, and associated with this there is considerable hyperæsthesia of the skin. The most tender place is usually in the thigh some little way below the anterior superior iliac spine. The hyperæsthesia may be on the inner side of the thigh. The surface may be so tender that the patient cannot submit to an ordinary examination of the hip, and calls out if the part be merely handled. The pain may run down the thigh to the knee, and be severe in the knee. This extended pain down the limb may be present without the hyperæsthesia just alluded to. The thigh is kept flexed a little on the pelvis, and there is some lordosis. The patient cannot bear the limb to be moved, and usually holds it with both hands when the bed is approached. At the same time there is fever and malaise, and the patient looks anxious and ill. The bowels may be regular and there may be no vomiting.

It is no matter of surprise if such a case be regarded as one of acute hip disease. The diagnosis, however, soon becomes apparent, and very often an abscess in the right iliac fossa makes its appearance, and directs attention to the real seat of disease. During the time when the features are not like those of acute hip trouble, the discovery of a vague swelling in the cæcal region does not of necessity clear up the diagnosis.

Perinephritic abscess.—If the abscess due to perityphlitis make its way into the connective tissue around the kidney it is of course actually perinephritic. If it be still intraperitoneal, however, it may be so placed in the region of the kidney as to suggest its origin in that gland. There are, moreover, certain possible symptoms in perityphlitis which may give rise to a suspicion of kidney mischief; these are a pain extending from the right side of the belly down to the bladder, or the external genitals, or the inner side of the thigh; and among the phenomena attending such pain may be frequency of micturition. The thigh may be kept drawn up. The patient may have passed gravel.

I have operated upon a case in which there had been severe attacks

of pain without either fever, vomiting, or apparent swelling in the cæcal region; but each attack was attended by intense hyperæsthesia of the skin over the renal region. I discovered a strictured appendix full of muco-pus, situated entirely behind the cæcum and adherent to the iliac fascia. The cæcum was above its usual level.

Psoas abscess. — The difficulty of distinguishing between a perityphlitic and a psoas abscess may be considerable. The spinal abscess may occupy the iliac fossa on the one hand, and the abscess from the bowel may exactly occupy the position of the psoas muscle on the other. In the latter instance there may be retraction of the psoas muscle, the thigh being drawn up. There may be great pain in straightening the thigh, and pain in the back.

As a rule the circumstances of the case are sufficiently distinguishing. In the spinal abscess there is no history of sudden abdominal pain nor of intestinal trouble. In the perityphlitic abscess there is no associated rigidity and deformity of the spine.

These distinguishing points are, however, not always clear, and I have met with cases in which a right diagnosis was very hard to attain.

Other conditions.—Troubles in the appendix may be confused on more or less reasonable grounds with inflammation of the ovary, or with inflammation and suppuration of the Fallopian tube. Such confusion is the more likely in those examples of perityphlitis in which menstruation appears in some way to be associated with the attack (page 919).

It is said that a twisted floating kidney and a pelvic hæmatocele have been the causes of errors in diagnosis in respect of perityphlitis.

I have known a distended bladder in an elderly lady with undoubted perityphlitis to be mistaken for peritoneal effusion. There was no suspicion that the bladder was not empty, and the swelling which already existed in the right iliac fossa was supposed to be extending across the middle line; the dulness in the cæcal region being continuous with that due to the distended bladder. It should be remembered that retention of urine is common in these cases, and that the peritoneal effusion rarely extends beyond the median line.

Symptoms not unlike those of chronic or relapsing typhlitis may be due to extensive adhesions in the cæcal region. Such adhesions may cause attacks of colic and temporary obstruction which bear a resemblance to perityphlitic attacks. Very commonly these adhesions have been induced by an actual perityphlitis of ancient date; in one such case, which I explored by operation, I found numerous adhesions involving the ileum and omentum; but the appendix was converted into an inert fibrous cord.

COMPLICATIONS.—*Pylephlebitis and hepatic abscess.*—This is, fortunately, by no means a common complication. Fitz notes 11 instances of mischief in the liver in 257 cases of perityphlitis, but, generally speaking, the proportion is very much too high. I should think that the

probability of such a complication may be more nearly represented by 0·5 per cent. The trouble, when it occurs, is due to an infective thrombosis which involves the superior mesenteric and portal veins and spreads from the seat of disease to the liver. The liver becomes dotted over with minute specks of necrosed tissue, and later with multiple small abscesses. It is a species of pyæmia limited to the portal system. A few examples have been recorded in which the infection extended beyond the liver and led to abscesses on the brain and lung.

The thrombosis may implicate the splenic vein, and an abscess of the spleen may result. It would appear that an embolus may be carried from the vessels of the diseased appendix, and may lead to a solitary abscess of the liver, without any thrombosis of large radicles of the portal vein. In such instances the pus on the abscess is usually very fœtid.

Dr. Payne has shown that infective thrombosis of the superior mesenteric and portal veins may result from ulceration of the mucous membrane of the appendix without any peritonitis, that is, without any signs of perityphlitis. Dr. Janeway has recorded a similar case. I too have met with such a case clinically, and mentioned it on page 913.

The symptoms of pylephlebitis are very marked, and can scarcely be misinterpreted.

I have opened the abdomen by operation in a case of pylephlebitis following typhlitis in a girl of fifteen. The symptoms were very pronounced, and the patient's condition apparently hopeless. The liver surface was dotted over with the innumerable yellow specks described by pathologists. The patient made a good recovery. The case is recorded in my Lettsomian Lectures on "Peritonitis," *Brit. Med. Journ.* February 3, 1894.

Pleurisy or pneumonia, mostly of the right side, may be expected in about 1 per cent of the cases of perityphlitis. These lung complications are therefore not so common as in other forms of peritonitis. In acute peritonitis considered generally lung complications may be expected in about 17 per cent of the cases.

Pulmonary embolism from thrombosis of the pelvic veins has been recorded as occurring on the fifth day after operation of perityphlitis (Roux).

Thrombosis of the iliac vein on the right side may occur. This also has led to pulmonary embolism. Œdema of the right leg may occur without thrombosis, and be due to the pressure upon the vein of the inflammatory exudation about the cæcum. I have met with two cases in which œdema of the left leg appeared after perityphlitis. No explanation of this symptom was forthcoming; it did not last long.

Fatal bleeding has occurred from a perityphlitic abscess after it has been opened. The hæmorrhage is from the deep circumflex artery and from the iliac vessels (Bull).

Intestinal obstruction of an acute character may be produced by strangulation, by adhesions, or even by an adherent appendix itself. It has resulted also from the kinking of an adherent coil. After the evacuation

of a perityphlitic abscess subacute obstruction may be due to kinking of the bowel, which, being adherent to the inflamed mass, was bent upon itself when the mass contracted after operation. The gut involved in these cases is usually the sigmoid flexure.

Symptoms of chronic, partial, or occasional intestinal obstruction, extending over months or years, may follow from adhesions which implicate the coils of the lower ileum. In one case in which I operated the coils of the small intestine were matted together in inextricable confusion. The patient had been liable for years to attacks of colic with some sickness. There was constant uneasiness in the right iliac fossa, with rumbling and gurgling in that situation, and occasional evidence of enlarged coils of bowel in uneasy movement.

Inflammation of the parotid gland has been reported as a rare complication. It is most common about the end of the second week.

I have met with one instance in which *acute epididymitis* occurred on the third day of a severe attack of perityphlitis. There was positive evidence that the urethra was free from disease at the time.

Fæcal fistula.—A fæcal fistula may result after a perityphlitic abscess. It is most usually associated with a sinus in the skin in the right iliac region, and communicates with the cæcum.

The fæcal sinus has been in the loin, at the umbilicus, and even at the bottom of a hernial sac. The fistulous tract may be internal, and extend between the cæcum and the rectum or the bladder. A bladder fistula is very rare.

The fæcal fistulas now under discussion are apt to be long-abiding. They may continue for months or years, but in general terms it may be said that they show a disposition to close, and that in the course of time the great majority do close. On the other hand, they are exceedingly difficult to deal with by operation.

Hypochondriasis.—I have said that a subject of relapsing perityphlitis—usually a man—will be met with now and then who has become quite melancholic on the subject of his disease, or at least to be hypochondriacal upon the state of his intestines.

Such patients devote themselves with a morbid ardour to the examination of their disorders, they are the subjects of remarkable and inexplicable symptoms, and are the recipients of an incredible variety of drugs.

Mortality.—The precise risk to life of perityphlitis is a little difficult to estimate. The statistics available are hospital statistics, and the cases of slight degree do not find their way into hospital wards; whereas the serious and the neglected cases are sure to be very fully represented. It is probable that the mortality of perityphlitis, taking all phases of the disease together—the most trifling attacks with the most serious—is about 5 per cent.

Hospital statistics from which the slight attacks have nearly all to be eliminated give a much higher death-rate. Dr. Hawkins deals with the circumstances of 264 cases admitted into St. Thomas's Hospital. Of

this number 37 died, a mortality of 14 per cent. This would probably be the death-rate of what may be termed a definite or sharp attack. Furbringer, dealing with 120 hospital cases, gives the mortality at 10 per cent. In the London Hospital the death-rate, as estimated three years ago, was over 20 per cent. Fowler, dealing with 99 cases at the Middlesex Hospital, records 15 deaths. These figures always appear high to the general practitioner, who must see a very large number of trifling attacks in which recovery takes place in a day or so, and cannot imagine that such patients are subjected to a risk to their lives represented by 14 per cent. Guttman, taking the cases of all grades occurring in the Moabit Hospital, found only 5 deaths in 96 cases.

Even Dr. Hawkins' statistics show that, in what some would call the "ordinary" cases, recovery is apparently invariable; and it must be remembered that the trifling attacks are in great excess even of the ordinary attacks. (For Dr. Hawkins' statistics see page 900.)

Perityphlitis would appear to be the most fatal in the quite young and the quite old.

The common causes of death are diffuse peritonitis, collapse, septicæmia, exhaustion, and troubles arising from the abscess.

When an abscess forms in perityphlitis it is probable that the risk to life is at once raised to 30 per cent. Bull found the death-rate in 67 cases of abscess which were not treated surgically to be 48 per cent.

Fitz in his analysis of 176 fatal cases found that the day of death was as follows:—

	Per cent.
Death on the 2nd day	4
,, 3rd ,,	11
,, 4th ,,	7
,, 5th ,,	11
,, 6th ,,	9
,, 7th ,,	12
,, 8th ,,	12
,, 9th ,,	6
,, 10th ,,	4
11th to 20th ,,	7
In 4th to 8th week	8·5

It will be noticed—and the matter is important in connection with treatment—that only 4 per cent die within forty-eight hours, and only twenty-two per cent before the fifth day.

The highest death-rate comes between the seventh and eighth days.

TREATMENT.—This may be considered under the three following heads: (i.) The treatment during an attack; (ii.) The treatment between the attacks; and (iii.) Operative interference.

(i.) **The treatment during an attack.**—Absolute rest in bed is imperative: a hypodermic injection of morphia is given, and a warm fomentation is applied to the abdomen. In children the injection of morphia will be replaced by a suitable dose of tincture of opium or of chlorodyne. In adults the amount of morphia given must vary with the

intensity of the pain. One-sixth of a grain will often suffice. It may be repeated if necessary; but it should be made a special condition of the treatment that as little morphia as possible be given, and that it be employed only to relieve pain. So long as there is any vomiting no food should be given by the mouth. Should the vomiting be very obstinate—a most unusual feature—all feeding (such as it is) must be by the rectum. When the vomiting has subsided the food given should be fluid, administered hot, and in small quantities. Hot weak tea, hot milk and lime water, hot beef tea, represent the usual elements of the diet at the beginning of the trouble. Ice and iced drinks are to be avoided. As time goes on the diet may become more liberal, but must remain fluid for some time. As a rule milk is not suited for these cases; if it be given it should be first peptonised. Some malted foods, or toast soaked in tea or milk and water, represent the transition to a more solid diet. Solid food—in the sense in which the term is usually employed—should not be given until the tongue is cleaning and until the bowels have been opened and the temperature has declined. Too long perseverance with a purely fluid diet often leads to troublesome flatulence, yet the too early employment of solid food leads to dyspepsia, to abdominal pain, and often to a revival of the fever.

The most difficult problem in the early treatment of the attack is that of an aperient. In my opinion the safest line of conduct in this particular is as follows:—If the onset be mild a purgative should be given at once. Over and over again an attack has apparently been cut short by a promptly administered aperient. Certain of the subjects of a mild degree of relapsing perityphlitis assert that, as a rule, they can bring an attack to a successful issue if they can take an aperient in time. In attempting to promote an action of the bowels it is best to give an enema at once. Should it cause a thorough evacuation of the bowel no further measure is needed; should it fail—as is very often the case—a purgative should be given without delay. Castor oil can seldom be tolerated, and if not, one grain of calomel may (in an adult) be administered every hour until four or five grains have been taken; or two drachms of sulphate of soda may be taken every hour until the bowels act, or until some four or five doses have been taken. Supposing, on the other hand, that the onset of the attack be very acute and severe, the administration of an aperient at once cannot be entertained. Morphia is to be given, and as soon as the pain has subsided an enema may be employed. If it act it is well; but very often it causes increased pain and utterly fails. In any case it should not be repeated, and no aperient should be given. The main indication is to restore a condition of rest within the abdomen. In the quite acute cases no aperient is of any avail; as a rule, indeed, the drug is vomited at once. It is well, therefore, in such cases to wait a few days until the acute manifestations have subsided, and then on the fourth to the sixth day—if the case be progressing well—to make an attempt to relieve the bowel by an aperient, followed or assisted by an enema. When the bowels have once acted we

should endeavour to maintain this action every other day, if there be no indication to the contrary.

If, as the case advances, there be a distinct local swelling with definite dulness, if the local symptoms be prominent, the fever persists, and the general phenomena show no improvement, five to six leeches may be applied over the right iliac region. This measure very often has a magical effect. The precise time at which leeches may be applied must depend upon the individual case; I have used them with advantage at various periods between the third and the ninth days. It is needless to say that when once suppuration has occurred they are useless. In perityphlitis the onset of suppuration is often unaccountably delayed.

The local application of ice has been credited with good results by some.

The patient should remain in bed, and be kept upon a spare diet until all fever has subsided, and until the cæcal region is free from tenderness and swelling. Too early movement is very apt to cause a relapse.

As soon as the bowels are acting freely, salol may be given (in a powder) in 10-grain doses night and morning. β-Naphthol is preferred to salol as an "intestinal antiseptic" by some physicians.

Certain writers have claimed marvellous powers for sodium salicylate in perityphlitis. The drug is given in 20-grain doses every three hours, and is especially advocated by those who believe in the rheumatic or gouty origin of the affection.

The patient should not resume an active occupation until he is perfectly sound, as a relapse is not uncommon. I have known a relapse to follow indiscreet massage after an attack.

(ii.) **The treatment between the attacks.**—In order to do what can be done to prevent another attack, the following measures are to be advised:—

The digestion must, above all things, be attended to. If the teeth be defective all deficiencies must be made good. In many instances I have known a set of false teeth to bring a case of relapsing perityphlitis to a favourable ending, the patients having had no further attacks. Experience shows that the bolting of ill-masticated masses of food is a common exciting cause of perityphlitis. The meals must be taken at regular hours, the patient must eat slowly, and must rest after each meal. These recommendations should be insisted upon. Not a few attacks in active business men can be ascribed to a hurried lunch, which is eaten to-day at one o'clock and to-morrow at three.

The food must be carefully selected, must be simple and digestible, and of such a kind as to leave as little refuse as possible in the intestine. Milk and much animal food seldom appear to answer in these cases. There is no pathological evidence to support the advice that the patient should especially avoid substances, such as strawberries, figs, or currants, which contain minute seeds. It is essential that he should avoid articles of food which are likely to remain undigested, or to be retained and to decompose in the bowel.

The bowels should be made to act daily. Enemas and aperients must be given if needed. They should be avoided if possible. Much may be done by suitable dieting and easy exercise to encourage a normal action. Indigestion is frequently at the bottom of the constipation which occurs in these cases.

Massage of the abdomen appears in many cases to have a very admirable effect; partly, it may be, by promoting the absorption of inflammatory exudations, and partly by encouraging a normal action of the bowels. Exercise in the open air is in every way to be encouraged.

Salol or β-naphthol may be given night and morning for a month at a time. These drugs undoubtedly have some effect in arresting the process of decomposition in the bowel, a fact which is often demonstrated in patients with an artificial anus. The salol must be given in a powder (in cachets); when in the form of a tabloid the drug is very apt to pass through the intestine unchanged.

The patient who wishes to avoid another attack should abstain from violent exercise, such as hunting; and should avoid exposure to fatigue, damp, and cold.

(iii.) **Operative interference.**—This question may be discussed under two heads: (*a*) Operative interference during an acute attack; (*b*) Operative interference in relapsing perityphlitis.

(*a*) *Operative interference during an acute attack.*—The main feature in this treatment consists in the urgent advice that a free incision should be made down to the inflamed area as soon as there is evidence that suppuration has taken place. To this rule there is practically no exception. It is not always easy to tell when suppuration has taken place; the swelling may be large and tender, the temperature may be high, the symptoms may persist for some days, and yet the whole may end in complete resolution. Cases which begin very acutely may end in speedy recovery, while those which begin mildly may pass on to suppuration (*vide* p. 906).

If the swelling is marked and the part very tender, the fever high, and all the local symptoms persisting and perhaps increasing, the question of operation becomes a pressing one.

It may be laid down as a rough rule that the use of the knife will very seldom be called for before the fifth day. Indeed, I would venture to think that surgical interference before the fifth day should not be undertaken except in the presence of emphatic symptoms. Indeed, the great majority of the operations for perityphlitis are performed after the first week.

The treatment of this disease by rational and precise surgical methods is a matter of recent years. The older method of practically leaving the malady to itself, and of not opening the abscess until it was about to burst through the skin, may excuse the excessive enthusiasm of some modern surgeons who have gone to the other extreme, and advise the use of the knife without compromise and without delay. One writer on this subject compares the delay in operating in perityphlitis to a like delay in

dealing with strangulated hernia. That the author does not recognise the incongruity of his comparison is shown by the following heroic advice:—"After persevering for forty-eight hours with this treatment" (an original treatment by means of opium, saline purgatives, and massage under ether), "if relief is not obtained, I would at once resort to laparotomy." Another American surgeon considers "all cases of appendicitis as being imminently dangerous to life from the beginning of the attack." This conclusion is absolutely at variance with facts. This surgeon always operates within twenty-four hours if medical treatment fail. Another writer asserts that if operation be delayed until the sixth day 50 per cent of the patients die,—an assertion which is also totally opposed to facts.

It is urged in favour of early incision that a large number of recorded cases afford examples of an operation performed too late, or of a fatal issue which could have been averted by operation. Such cases exist without doubt. They afford an argument in support of earlier interference, but do not necessarily imply that the opening of the abdomen within the first twenty-four, or forty-eight, or seventy-two hours should be the routine treatment of a case of perityphlitis. The number of cases which undergo spontaneous cure form an overwhelming majority, and cannot be lost sight of; nor can the opening of the abdomen through the muscular parietes over the cæcum be regarded as a trifling procedure. Again, it is urged that certain cases have ended fatally within the first thirty-six hours by perforation into the general peritoneal cavity. Such cases are, however, exceedingly rare; they cannot be anticipated, and they are not difficult to recognise. In most of them the very first symptoms are those of perforative peritonitis. When such a case is met with, the abdomen should of course be opened at once, and the perforation dealt with as the practice is in dealing with other forms of perforative peritonitis. A case of death from perforation within thirty-six hours of the appearance of the symptoms of perityphlitis does not afford a legitimate argument for the routine performance of an operation within that period, even in the majority of the cases. It would be as wise to advise immediate operation in all cases of ulcer of the stomach as soon as the diagnosis is made, because some cases of ulcer of the stomach end in a fatal perforation. Some of the milder forms of perityphlitis—those which end early in resolution—may begin with quite acute symptoms, so that the severity of the symptoms alone is not a test of the need for early operation; although it is true that, in general terms, the more acute the manifestations the greater the need for prompt surgical measures.

Then, again, it is asserted that the abscess, if left, will burst into the peritoneal cavity and cause death, and that such a termination has been recorded when the abscess contained but an ounce or so of pus. This assertion, again, does not afford an unqualified argument in favour of the early use of the knife. As a rule the abscess makes its way through the abdominal parietes, and does not burst into the general serous cavity. Dr. Bull has shown that in sixty-seven cases of abscess the pus escaped

in twenty-eight instances through the belly wall, and that in only eight examples did it burst into the peritoneal cavity. Whilst it is true that small collections of pus have followed the latter course, it is also true that abscesses containing pints of matter have been successfully opened through the integuments some weeks after the commencement of the symptoms of perityphlitis.

In this relation it must be remarked also that a very small collection of pus is not easy to discover, and that in not a few recorded cases an early incision did not reveal the collection. It is true that the sooner retained pus can be let out the better, but that aphorism does not guarantee the harmlessness of speculative incisions in search of it; especially when the pus is within the abdomen, and in a region the anatomical features of which are liable to variation. Moreover, it is desirable that the matter should be well localised, and that the resulting adhesions should have connected the inflamed district directly with the parietes. The later the operation is delayed the more easily and directly can the pus be reached. If a very early incision become the rule, it will frequently happen that the matter cannot be reached without first opening the general peritoneal cavity, and cannot escape without first finding its way into that space.

Deeply-seated pus is slow to make its presence evident, and in the majority of cases the evidence will not be unequivocal before the fifth day. Should its presence be made clear before that period it is obvious that surgical interference should not be delayed; and it must also be allowed that urgency of symptoms may justify an exploratory incision before the arbitrarily fixed time is reached.

If on incision made pus is discovered, the abscess cavity must be freely drained and treated upon the usual surgical principles.

The use of the exploring needle—which has been much advocated by American surgeons—is to be very strongly condemned. This needle is thrust into the iliac region of the abdomen, often to the depth of three or four inches, is passed in different directions, and is sometimes introduced three or four times at one sitting. The object is to discover pus. In the first place, it may be pointed out that deep-seated pus, in sufficient quantity to demand surgical interference, may be diagnosed by other means; and that if an exploration must be made it would be safer to trust to a cautious incision than to a series of plunges made in the dark.

In the next place, the use of the needle is not free from risk. In the course of its employment it must be thrust now and then into the cæcum, and, if that part of the bowel be in a condition of ulceration, it is possible that the needle may induce a suppuration that previously did not exist. It has been shown that the use of the hollow needle in extreme tympanites is not unattended with risk, and it is not to be expected that the risk will be less when it is thrust into the inflamed and softened coat of an important part of the bowel.

Perityphlitis is often due to extreme distension of the appendix with mucus. An appendix so involved may reach the size of a walnut

This perityphlitis may end in complete resolution. A needle introduced during the acute period in such a case may very easily tap the distended septic process, allow a quantity of fœtid mucus to escape, and so bring about a suppuration which was by no means inevitable.

It would be unfair to draw conspicuous attention to some cases in which repeated puncturings gave no evidence of pus, but in which an abscess appeared some days later. Even when an abscess does exist, it must be remembered that its walls are often formed by the cæcum and some coils of ileum matted together, and into such an abscess (when small) a needle can scarcely be introduced without risk.

The needle, moreover, does not always appear to justify the arguments for its use. In one of Dr. Bull's cases the needle-punctures revealed nothing, but an incision made immediately afterwards evacuated no less than four ounces of pus. In other instances such large quantities of matter have been let out shortly after the presence of pus had been made evident by the needle, that we may well presume that the pus might have been detected by other and simpler means.

Into the surgical details involved in the actual treatment of the abscess I have not here to enter, except to say that the pus should be reached by the most direct route and by a free incision, that fæcal concretions should be gently sought for, and the appendix removed should it actually present itself. No deliberate and elaborate search should be made for it. The main indication is to open the abscess. The less the cavity is manipulated the better, and the bottom of a septic abscess is not the best place for carrying out the elaborate plastic operations advised by those who counsel the determined removal of the appendix in every case. Beyond a free opening and free drainage of the suppurating space the less done the better.

For the mortality of these measures see page 928.

(*b*) Operative interference in relapsing perityphlitis.—The treatment of cases of relapsing perityphlitis by removing the appendix during the period of quiescence offers, probably, more admirable results than are to be obtained in the treatment of any other form of the disease.

The circumstances which would justify an operation in these cases must be precisely defined, and it cannot be too emphatically stated that in a fair proportion of instances in which the trouble has relapsed no surgical interference is called for.

I am aware of many cases in which a patient has had three or more attacks of perityphlitis, and has then ceased to be troubled with any further outbreaks. In some examples of the relapsing form much can be done by the medical means already described, by diet, by attention to the bowels, and by placing the patient under conditions more favourable to a state of peace within the abdomen.

The operation simply consists in the removal of the offending organ—the appendix. I first proposed this operation (which should be carried out during a quiescent period) in 1887, in a paper read before the Royal Medical and Chirurgical Society. Since that date the pro-

cedure has been performed in a great number of cases, and not always, I venture to think, with proper discrimination.

The following are the more important circumstances which would justify an operation; and in all the cases with which I have dealt, one or other of the subjoined conditions has been present:—

(α) The attacks have been very numerous, although possibly slight. In some cases the patients had had twenty to thirty attacks.

(β) The attacks are increasing in frequency and severity.

(γ) The last attack has been so severe as to place the patient's life in considerable danger.

(δ) The constant relapses have reduced the patient to the condition of a chronic invalid, and have rendered him unfit to follow any occupation.

(ε) The patient is prevented from following a special employment unless he can consider himself free from the possibility of another attack.

(ζ) Owing to the persistence of certain local symptoms during the quiescent period there is a probability that a collection of pus exists in or about the appendix.

In the majority of the cases in which I have operated I have been able to make out the enlarged appendix after the acute symptoms had passed away.

It may be stated that the pain and distress involved by the operation will be less than that attending any but a slight attack, and that the risk of the procedure is less than that associated with an outbreak of perityphlitis considered generally. I have removed the appendix during the quiescent stage in over 150 cases of relapsing perityphlitis with only one death—a mortality of less than 1 per cent. The general mortality of an attack of perityphlitis cannot probably be less than 5 per cent (see page 928). In every one of the patients who have recovered from the operation the cure has been absolute and complete. The youngest patient upon whom I have performed the operation was seven years of age, and the oldest was sixty. Ventral hernia is uncommon after this particular laparotomy.

The operation may prove to be very simple; on the other hand, it may prove to be exceedingly difficult. In one or two instances the removal of the appendix—on account of the nature of the adhesions—was found to be impossible.

The death-rate after removal of the appendix during a period of quiescence has been in my experience 1 per cent. In Dr. Hawkins' 38 cases of suppurative perityphlitis, 27 operations were performed. The abscess was in each case opened and drained. The appendix was not removed. Of this number four died, a mortality of 14·8 per cent. In six cases no operations were performed, all the patients died; in the remaining five cases the abscess burst into the bowel, with recovery in each instance.

FREDERICK TREVES.

REFERENCES

1. BLACKADDER. *Edin. Med and Surg. Journ.* 1824.—2. BRISTOWE. Reynolds' *System of Medicine*, vol. iii. p. 121.—3. BRYANT. *Brit. Med. Jour.* vol. ii. 1884.—4. BURNE. *Med.-Chir. Trans.* 1837.—5. DUFOUR. *Bull. Soc. Anat.* 1852.—6. EARLE. *Chicago Med. Journ.* Feb. 1876.—7. EINHORN. *Munch. med. Woch.* 1891, p. 121.—8. FENWICK and DODWELL. *Lancet*, July 1892.—8*a*. FÉRÉ. *Progrès méd.* 1877.—9. FITZ. *Amer. Jour. of the Med. Sciences.*—10. *Idem. Boston Medical and Surgical Journal*, 1890.—11. HABERSHON. *Diseases of the Abdomen*, p. 414.—12. HARLEY. *St. Thomas' Hospital Reports*, 1881, p. 128.—12*a*. HAWKINS, H. P. *Diseases of the Vermiform Appendix.* London, 1895.—13. HAY, ALEX. *Brit. Med. Journ.* June 30, 1894.—14. HODENPYL. *Annals of Surgery*, Jan. 1894.—15. JANEWAY. *Trans. Amer. Assoc. of Physicians*, 1886.—16. KELYNACK. *The Pathology of the Vermiform Appendix*, 1893, p. 112.—17. *Ibid.* p. 107.—18. LANG, OTTO. *Korrespond. für Schweitzer Arzte*, 1892, No. 19.—19. LEWIS. *New York Journ. of Med.* 1856.—20. M'CALLUM. *Med. News U.S.A.* Dec. 17, 1892.—21. MELIER. *Journ. gén. de méd.* 1827.—22. MOORE, NORMAN. *Trans. Path. Soc.* 1883, p. 113.—23. MORTON. *Lancet*, April 14, 1894, p. 971.—24. MURCHISON. *Continued Fevers*, 2nd ed. p. 623; *Trans. Path. Soc.* 1866, p. 127.—25. PAYNE. *Trans. Path. Soc.* 1870.—26. PILCHER. *Annals of Surgery*, 1893.—27. PORTER. *Boston Med. and Surg. Jour.* Dec. 25, 1890.—28. *Idem. Amer. Journ. of Med. Sciences*, 1893.—29. RANSOM. *Med.-Chir. Trans.* 1892.—30. RIBBERT. *Virchow's Archiv*, 1893.—31. TALAMON. *Méd. moderne*, June 19.—32. TAVEL and LANZ. *Ueber die Aetiologie der Peritonitis.* Basel, 1893.—33. WEIR. *Med. Record*, Jan. 10, 1880.—34. WILKS. Fagge's *Medicine*, vol. ii. p. 174.—35. YEO, BURNEY. *Brit. Med. Journ.* vol. i. 1894.

F. T.

DISEASES OF THE COLON

I. Simple colitis. — By this is meant a catarrhal inflammation of the large intestine, comparable to gastritis or to bronchitis. Recently I had an opportunity of showing this state in the case of a man who came into the hospital with severe diarrhœa. As there was some uncertainty whether an ulcer could be felt high up in the rectum, an anæsthetic was given and a rectal examination was made with a speculum. As far up as we could see, and in all probability much farther, the mucous membrane was intensely injected, of a bright red colour, swollen and secreting a thick mucus; but no ulcer was visible. The patient quickly recovered under appropriate treatment.

Then, again, we have post-mortem evidence of a more severe condition. For instance, Wilks and Moxon mention a "case attended by discharge of mucus and blood where, after death, the whole internal surface of the colon presented a highly vascular, soft, red surface covered with tenacious mucus or adherent lymph, and here and there showing a few minute points of ulceration. The coats also were much swollen by exudation into the mucous and submucous tissues." A very well-marked instance was admitted into Guy's Hospital under Dr. Perry on April 19th, 1891. The patient, a woman aged 36, was in her seventh month of pregnancy. For the fortnight previous to April 13th she was slightly sick. At the

latter date the vomiting became worse. Her face was sunken, the rectum was empty, the bowels were constipated. She died soon after admission. For the last few hours of life she had diarrhœa. The whole colon was inflamed, the mucous membrane being thick, sodden, and deep purple; no ulceration and no membranous exudation could be seen. The liver was fatty.

There are two varieties of simple colitis; one, which is unassociated with any other serious malady, may be called *acute primary colitis*, to distinguish it from the results of direct injury or extension from neighbouring parts, and from colitis as associated with constitutional diseases such as Bright's disease. Acute primary colitis has been called mucous colitis; but as this name has been also used to describe what I shall call membranous colitis, it is liable to lead to confusion. Of several cases that have been under my own care, I may quote the three following as being good examples of acute primary colitis:—

A lady aged 25, under the care of Dr. Lacy of Woolwich, was suddenly seized with pain in the abdomen; this was quickly followed by diarrhœa, which became very frequent; the motions consisted chiefly of mucus, but they contained some bright blood. Pressure over the transverse and ascending colon caused some pain. The tongue was covered with a very white fur. There were loss of appetite and nausea, but no vomiting. The temperature varied between 99° and 101°.

The second case was a man aged 30, who suddenly one morning was seized with oft-repeated severe diarrhœa. The motions at first consisted of nothing but mucus and blood. In the course of five days the diarrhœa slowly ceased and the motions contained more fæcal matter. His temperature gradually mounted, till on the fifth day it was 104°. Then there followed marked constipation. On some occasions he had much severe griping pain, chiefly along the course of the large intestine when the bowels were open. There was also abdominal tenderness. He recovered completely.

The third case was under the care of Dr. Payne of Witham. The patient, a man aged 38, was suddenly seized with diarrhœa; the motions contained much mucus and blood, and the microscope showed the presence of red blood cells, leucocytes, and triple phosphate crystals. He had considerable griping pain, abdominal tenderness along the colon, and loss of appetite. His tongue was covered with a white fur. For three months this train of symptoms continued and he lost 20 pounds in weight. He then consented to take to his bed, and from that time he began to improve.

Symptoms.—The main symptom of this disease is a diarrhœa, which may come on suddenly; there is much mucus in the stools, and often blood also, even in considerable quantities. At first there may be absolutely no fæcal matter; but as the patient improves the motions contain more fæces and less mucus. The blood is mostly fluid, and but little changed either to the naked eye or the microscope; we infer that it has but recently left the vessels, and has come, therefore, from the large intestine. Often the mucus is in little lumps

Triple phosphate crystals may be seen on microscopical examination; and, more rarely, oxalate of lime, cholesterin, and Charcot's crystals. In exceptional cases the motions have been described as frothy. The bowels may be opened many times a day; tenesmus is often present, but it is not by any means always a striking feature. A rectal examination reveals nothing abnormal, except that sometimes the mucous membrane feels a little rough. Some patients pass a considerable amount of wind from the anus, and complain of rumbling or of borborygmi within the abdomen. Abdominal pain is a very common symptom, it comes on in paroxysms, often associated with defæcation, but it bears no relation to food; in this respect it stands in contrast to many gastric pains. It is of a griping character, often very severe; and it nearly always follows the course of the colon. Between the attacks the patient may be free, but he sometimes still complains of a dull pain. Abdominal tenderness is usually present; a very common seat for it is over the sigmoid flexure, but the whole colon, or even the whole abdomen, may be tender. According to Habershon, some such patients suffer from irritability of the bladder and dysmenorrhœa.

As we have seen from the cases described, there may be considerable pyrexia; and it is noteworthy that the temperature may remain raised even when the patient is much collapsed from diarrhœa and loss of blood. The pulse is rapid, and, in a severe case, small, soft, and running. The abdomen usually moves with respiration, and is not commonly much distended. Some patients vomit, there may be considerable nausea and loss of appetite, thirst may be a cause of complaint, and if the diarrhœa is unchecked, anæmia and loss of flesh may follow. The tongue is furred, and it is very important to remember that the colour of the fur is almost pure white, whereas in many diseases with which colitis may be confounded it is a brownish or yellowish white. The fur is often universal, but the tip and edges may be clean. As a rule these patients suffer much from mental depression; they exaggerate trifles and take a gloomy view of life. In some patients the neurotic element is so marked that it forms the chief feature of the case; we are forcibly reminded of the neurotic dyspeptic invalid. In my experience this neurotic variety is more common in men than in women. Such, when they come to see the doctor, frequently bring with them a written statement containing a minute account of all the symptoms, and often a careful description of the motions, and are valetudinarian in aspect and behaviour. Such cases are often very chronic, and they are most difficult to cure.

Various amœbæ, infusoria, and other micro-organisms have been described in the motions of diarrhœa; they may exist in cases of colitis, and may perhaps be the cause of the disease. References to the literature of this subject are given by Eichhorst.

The reader must not suppose that simple colitis is always so serious as the above description might lead us to suspect; but the difference is only one of degree. The majority of cases are mild, when the symptoms are all less intense; and probably many cases of slight diarrhœa,

which it is difficult to classify, are really instances of mild colitis. On t other hand, the diarrhœa may be uncontrollable, and the patient may d from exhaustion. Once I saw a man who, when convalescent from a sev and prolonged attack, died with all the symptoms of pulmonary embolis

The *treatment* should be on the same general principles as tha diarrhœa (p. 773). The patient must be kept absolutely in bed until t diarrhœa has stopped, the motions are well formed, blood is no lon passed, and the temperature is normal; and longer still if he has ha severe attack. Warmth to the abdomen is very desirable. He sho consume nothing but milk, and should not take more than two fl ounces at a time. The total daily amount will depend upon the ac ness of the attack and the general condition of the patient; usually he take from two to three pints a day. A good way of checking the diarr is to give some laudanum with fifteen or twenty grains of carbonate bismuth suspended in some mucilage every four hours; the compo kino powder is also very valuable: if these drugs fail, a starch and opi enema will often succeed. Should opium for any reason be contra-indica compound catechu powder is very useful. I have sometimes t washing out the large bowel with a tepid saturated solution of bor acid, but I do not think there is much advantage in this. All astring drugs should be omitted as soon as possible, for the constipation t follows colitis is usually very troublesome. It is best relieved by a re injection of six or eight ounces of warm olive oil or a drachm of glycer A long holiday among new and interesting surroundings greatly aids convalescence in the neurotic cases.

Diagnosis.—This, as a rule, does not present much difficulty. disease is known from ulcerative colitis by its sudden onset, by the la amount of mucus in the motions, by the fact that blood appears earl the case, and also that under appropriate treatment it generally yie In England acute dysentery is hardly likely to cause much confusi but in any case the "meat washing" character of the stools, the burn pain in the rectum, the intense tenesmus, and the constant desire to to stool even when nothing is passed, should prevent a mistake. Ne theless, in England we often see severe cases of diarrhœa in persons have had dysentery abroad; and I think that persons who have dysentery are particularly liable to colitis. I have seen some extren bad cases, but under very strict treatment for the colitis the patients recovered. The character of the motions and the sudden onse diarrhœa distinguish colitis from enterica. It should never be forgo that malignant disease of the bowels often presents itself in such a g as to lead to errors of diagnosis. I have known a case diagnose colitis turn out to be chronic arsenical poisoning.

The *prognosis* is, as a rule, good. In recent acute cases, except in rare instances in which they are fatal, the patient recovers quickly; in cases that have been allowed to become chronic—and often the dis has lasted many months before it is seriously taken in hand—the pati require rest in bed and milk diet for many weeks.

Secondary colitis, by which I mean that which occurs either by extension from neighbouring parts, as a result of direct irritation, or in association with some grave morbid condition, is occasionally seen in the post-mortem room. Among the records of autopsies at Guy's I find the following varieties :—

i. *Colitis due to direct extension.*—I made a post-mortem examination on a woman who died of swallowing a large quantity of sulphuric acid. All along the greater curvature the whole thickness of the stomach was gangrenous, and the transverse colon was red and black on its exterior, and acutely inflamed on its inner surface. The stomach was in contact with the colon. The small intestine was unaffected, so it appeared that the effect of the acid had spread through the walls of the stomach to the transverse colon (36).

ii. *Colitis due to direct irritation.*—There is in the museum at Guy's Hospital the colon of a man who died in 1827 with severe diarrhœa, induced by large doses of purgatives given to overcome severe constipation. The colon is black, gangrenous and sloughy. Also it may be inflamed if corrosive sublimate or arsenic be given by the mouth, even when the small intestine is unaffected ; probably because the poison, after having been absorbed higher up, is excreted into the colon.

iii. There is a form of *acute colitis associated with septic or pyæmic conditions.* Of the five following examples, the first three are instances of an early stage of the process. A woman, aged 40, was admitted under Mr. Davies Colley in 1883 with chronic cystitis and suppurative pyelitis. "Rather intense colitis, the mucous membrane being rough, granular, ecchymosed, and swollen," was found. In 1886 a girl was admitted for an extensive burn. For some time she did well, but three days before death she was seized with vomiting and diarrhœa. There was pus in the right knee, the colon was vascular in patches, the solitary glands were enlarged, and Dr. Goodhart thought the condition one of early inflammation. In the same year a girl died from cancrum oris. The colon was injected, thickened, and covered with flakes of lymph. Also in 1886 a boy was admitted for compound comminuted fracture of the right tibia and fibula. Pyæmia, with pus in several joints, appeared, and he died a fortnight after admission. The mucous membrane of the rectum was rough, thickened, and vascular, with numerous minute ulcers and patches of yellow lymph. Higher up in the colon there were similar but less severe patches. There was a large patch in the cæcum and many sloughs in the colon. Dr. Goodhart remarks that the kidneys were exactly like some I described in the 26th vol. of the *Trans. Path. Soc.* ; that is to say, scattered throughout the organs were a number of patches consisting of an outer red zone, then a yellowish zone, and most centrally a brown area. A still more extreme colitis occurred in the case of a woman aged 28, who was admitted under Dr. Galabin in 1888 with pelvic abscesses and peritonitis from gonorrhœa. She had mitral and tricuspid stenosis, and died from exhaustion. In the cæcum and for two feet above it the mucous membrane was sloughing ; and from beyond

this to the anus the colon was acutely inflamed, its walls were œdematou and thickened, and its mucous surface was grayish yellow with patches o submucous hæmorrhage. The mucous membrane was separating in shreds in many places.

It will be noticed that in only two cases was there pus in the joints The other three had no remote pyæmic lesions. In no case is it possible to fix the duration of the colitis. In one vomiting and diarrhœa are mentioned. I do not think, however, that we can say much about the symptoms, for the importance of each of the cases was in other respects so great that it is quite likely that diarrhœa and vomiting, even if they were present, may not have been recorded. It is noteworthy that, although this is a rare condition, three cases occurred in Guy's in 1886.

iv. *Acute colitis* sometimes occurs *in association with Bright's disease*, but it is certainly unusual. The following case occurred in 1888:—A man aged 59, was admitted under Dr. Pye-Smith. He had chronic Bright's disease. For the last few days of his life he passed large quantities of mucus. At the post-mortem the surface of the colon down to the rectum was covered with mucus and catarrhal products. Another case was that of a man, aged 31, who had gout, granular kidneys, and mitral stenosis. During life there was much bloody diarrhœa. After death, in the lower part of the descending colon there was injection of the mucous membrane and a small quite superficial ulcer; near the rectum was another patch of injection and another small ulcer. The Guy's Hospital Museum contains three specimens of colitis in Bright's disease. The first was from a man, aged 32, who had large white kidneys. The colon happened to be sacculated; it was thickened, and had many sloughs on its mucous membrane; they were for the most part on the prominences between the sacculi. The second was from a man, aged 45, who also had tubal nephritis; the colon was inflamed, sloughing, and in parts black and gangrenous. The third was from a man, aged 25, who had tubal and interstitial changes in his kidneys. Here also sloughs were present in the colon, and there was superficial ulceration. Sometimes the colitis of Bright's disease is associated with acute inflammation of the small intestine, as in an instance recorded by Dr. Goodhart (22).

Perhaps some of these cases should be regarded as an early stage of ulcerative colitis, which, as I shall presently show, is often associated with chronic Bright's disease. Acute colitis is so rare a complication of Bright's disease that very little can be said of the symptoms. It is noteworthy however, that in all the cases there was severe diarrhœa; this, however, is a symptom of uræmia, and moreover purgatives are ordered for many persons suffering from Bright's disease. Further, very little stress can be laid upon hæmorrhage, as persons with Bright's disease may bleed anywhere.

v. *Colitis is associated with pneumonia* in extremely rare cases; two specimens of this are to be found in our museum. One was taken from a man, aged 20, who was admitted for pneumonia; his febrile symptoms lessened, but severe diarrhœa came on, he sank into a typhoid condition

and died. The whole of the large intestine was in a state of acute inflammation, and showed a few small rounded ulcers also. In the second case, on the eleventh day of the illness, diarrhœa set in, and six days later the patient died. In this case the colitis had proceeded to the stage of the formation of shallow ulcers. That the pneumococcus can affect many parts of the body is proved by the discovery of it in the meninges, in pleural effusions (48), in pericardial effusion, and on the endocardium in malignant endocarditis. Perhaps, therefore, it may directly affect the colon. It should be remembered that it is possible that some of the specimens of colitis found in the bodies of persons dead of septicæmia, Bright's disease, or pneumonia, may represent early stages of the membranous colitis which may complicate these diseases.

vi. If *the colon is lardaceous* it may, like the rest of the bowel affected with lardaceous disease, become, in very exceptional instances, the seat of acute inflammation.

II. Membranous colitis.—An inflammation of the colon leading to the production of a membrane is not very common; in proof of this I may mention that only a few lines are devoted to it in the edition of Eulenberg's *Real-encyklopädie* now appearing. Two distinct varieties exist: one which might be termed dyspeptic membranous colitis; and another which is always associated with some other grave condition, or is due to direct injury, and might therefore be called secondary membranous colitis. It is not usual to include under the heading of membranous colitis those cases of constipation in which when the bowels are open shreds of coherent mucus are passed with or without the hard masses of fæcal matter. Such cases are common enough.

Dyspeptic membranous colitis may be so called because the sufferers from it complain much of dyspepsia, and they pass membranes from the anus. Many other names have been used; a list of them will be found in an article by Dr. Light, who terms the condition desquamative enteritis; but it seems to me that this phrase fails to express that distinct membranes are passed, and that the chief seat of the disease is probably the colon. Dr. Light gives an excellent bibliography; one of the best collection of cases is that by Da Costa. Some German references will be found in Eulenberg's *Real-encyklopädie*.

The patients are usually over 20 years of age; the disease is commoner in women than in men, and in private than in hospital practice. It is a rare malady, but no doubt is often overlooked. In children it is excessively rare, for Edwards found that out of 111 cases only 6 were under the age of 10. The distinguishing feature of it is that membranes, which the patient usually calls skins, are passed from the anus. One patient whom I saw thought he was passing pieces of tapeworm; and this gives a very good notion of the usual consistence and colour of the membrane, which, however, may be brown from fæcal staining. In an extreme case it forms a complete tubular cast of the intestine, usually from one to six inches long; but such tubes have been known to be several feet in length, and sufficiently thick and tenacious to admit

of their being held up. The wall is occasionally laminated, and sm pieces of fæces may be detected between the laminæ, showing that th have been laid down successively. Fæcal matter may also be found i the interior of the tubes, the diameter of which may be anything up t 1½ inch; and the thickness of wall varies from an extreme tenuity to a thickness of one-fourth of an inch; the end may be well defined, but ofte it shades off into a soft, transparent, gelatinous material. The tubes ma be marked, apparently, by the valvulæ conniventes, or they may have a sacculated appearance, indicating their origin in the colon; their outer surface is smooth, the inner is more rough, probably from the attrition of the fæces. Either with or without the tubes shreds of all shapes and size may be passed, but in all essential characteristics they are the same as the tubes. Often several pieces of membrane, when passed, are rolled up in a solid ball. Under the microscope the membranes are transparent and structureless; embedded in them may be seen minute fragments of u digested food and fæces, some cells, free nuclei, putrefactive micro organisms, phosphates, and cholesterin crystals. The cells are apparent the epithelial cells of the large intestine which have undergone fatty degeneration. Groups of them may be seen compressed together in rows which suggest that after having multiplied they have been thrown off a have become embedded in the membrane. When the inner surface magnified it appears to be reticulate, and presents at regular interv depressions or even perforations which clearly correspond to the mouth of Lieberkuhn's crypts. Often the cells previously described are group around these openings in such a way as to show that the lining of follicle had been cast off and become incorporated in the membrane. Dr. Goodhart's case the pits were much larger than healthy follicles, fr which it would appear that the follicles had been distended.

Chemically the membrane consists of albumin; it contains no fibri

The reader who wishes to picture to himself a patient suffering fr this disease should have in his mind a chronic dyspeptic woman o depressed turn of mind and liable to constipation; though the mala may also occur in the busy, energetic, nervous subject. The *symptoms* the chronic dyspepsia are that the patients are poor eaters, believing t first this and then that article of food disagrees with them, so that th diet soon becomes very limited in choice and quantity; they are usu anæmic, thin, and complain of the cold; the tongue is pale and a little furr and the bowels are frequently rather constipated; but the constipatio occasionally alternated with mild diarrhœa. Some patients suffer from irritable bladder, and may pass much mucus in the urine. Da Co mentions a sense of rawness and even actual tenderness in the abdo as occasional symptoms, and he states also that sufferers from this dis are very liable to boils. They are often taciturn, they rarely have buoy spirits, and not infrequently suffer from great depression of mind, tak a gloomy view of life and exaggerating the importance of trifles. Th dyspeptic symptoms and this frame of mind are more or less consta with the patient, but there are exacerbations from time to time, du

which there is much abdominal pain, usually griping, generally somewhere in the course of the colon, and often coming on a long while after food; flatulence is troublesome, the loss of appetite and constipation are still more marked; the patient may complain of nausea, or suffer from vomiting; the tongue is more furred, and may be rather red; and the mental depression and feeling of general weariness are very pronounced. After this state of things has gone on for about a week the patient notices that the "skins" are passed by the bowel. In a mild case this lasts for a few days only, during which the dyspeptic symptoms may be less marked; she is then restored to her usual feeble health till another attack comes on; but in more severe cases the passage of membranes and the other symptoms continue for weeks or months, and much bright blood, which has clearly come from low down in the bowel, may be seen. In fact, sometimes when the bowels are open nothing but blood and membranes are passed, as in a patient I recently saw with Mr. F. K. Holman, who for weeks passed blood and membranes and very little fæcal matter. He had complete loathing of food, and although the passage of the membranes and blood stopped, by the time this happened he was so weak that he seemed incapable of rallying, and gradually sank and died. At the autopsy the colon was found to be considerably dilated, and its walls thin and atrophied, showing here and there a few patches of congestion. Otherwise all the viscera were healthy. Also in another fatal case I saw, although membranes were passed shortly before death, very few were found at the autopsy. These two fatal cases seem to show that the membranes are passed soon after they are formed. The membranes in the motion are often compressed into a tight ball about the size of a nutmeg; and when fæcal matter is passed it is in the form of a few small, hard, scybalous masses with pieces of membrane between them; even if ordinary fæces do come away, the first part of the motion usually consists of membranes only, or of the thick, transparent, tenacious, jelly-like material that occasionally replaces them: in Dr. Shingleton Smith's case, however (43), the fæces sometimes preceded the membranes. While passing them the patient is, for the most part, unusually constipated, and often takes enormous quantities of purgatives, but occasionally the bowels may be opened two or three times a day. The act of defæcation is often excessively painful. I have known a patient sit on the water-closet for a couple of hours suffering from agonising griping pain before a motion consisting of enough blood and mucus to full a half-pint measure was passed. She often used a morphia subcutaneous injection immediately before defæcation, and twice she fainted in the water-closet from the severity of the pain. A rectal examination reveals nothing abnormal. The abdominal griping pain is usually less after the bowels have been opened, and for a few hours the patient may feel a trifle better; but otherwise, unless the case be mild, the general symptoms present no such remissions. Dr. Goodhart's patient complained of feeling cold, and sometimes had rigors when the bowels were relieved. One patient under my own observation passed a moderate amount of uric acid gravel. Dr. Light

says that an excess of urates may be present, and that the subjects of this disease are liable to urticaria. If women, they often have amenorrhœa and pelvic troubles. With these exceptions the functions of the body are usually normal. The cases vary very much in severity. One patient I see occasionally has an attack, lasting about a week, on the average once a year. In the intervals she has fair health. On the other hand, many patients pass these membranes for months, or even very many years: the while they are perfectly wretched from chronic dyspepsia, they waste, become extremely anæmic, live in constant dread of defæcation, and spend all their money in travelling from health resort to health resort.

Between the attacks the patient should in all respects lead as healthy a life as possible. She should take plenty of outdoor exercise of a kind to interest her and take her out of herself; riding or playing golf are infinitely preferable to dull, solitary walks taken merely for the sake of the exercise. The diet should be ample; most of these patients decline first one article of food and then another, until at last their dietary is so restricted that they are not only underfed, but their digestive powers are over-taxed in some particular direction. An obviously indigestible dietary, such as one consisting largely of rich-made dishes, or an undue preponderance of sweets, should, of course, be avoided; but the patient should partake of any ordinary food that is put before her. It should be well cooked, and the meals should be made as tempting as possible to the slender appetite. Even if it be an effort, she must eat well. She should have her meals at regular times, and should go to bed early, and always have eight hours sleep. As these patients are always worse if they are idle, she should have some occupation that will constantly employ her mind. Purgatives should be avoided, for they are particularly liable to cause indigestion; the bowels should be kept open by going to the water-closet regularly at the same time every day, by healthy living, and, if necessary, by a light abdominal massage before rising in the morning. No drug is known to be of certain benefit, but Da Costa found iron useful. My experience of it in one case was that it made matters worse by the indigestion and constipation that it caused. A holiday and change of scene to some bracing place as Switzerland or Norway, or a yachting cruise, is often of the greatest benefit. Indeed, these patients derive much benefit from such a holiday, even when they are not passing membranes. During the period in which they are passed, it is particularly important not to give purgatives by the mouth; but, if the constipation be extreme and the griping pain be very severe, considerable relief may be afforded by a rectal injection of a drachm of glycerine, or half a pint of warm or soap and water. For a severe case, rest in bed, morphia injected hypodermically, and the application of hot fomentations to the abdomen may be necessary; but opiates should not be prescribed unless they are absolutely necessary; for not only do they increase the constipation, but sufferers from membranous colitis are just the sort of people who become addicted to an excessive use of these agents. In a fatal case which I saw with Mr. Holman, injections of boracic acid, which

strongly recommended by Brunner (7), were thrown high up the rectum, the patient lying on the left side with the thighs elevated; but as this was tried when the passage of membranes was lessening, it is difficult to say how far it was beneficial. In other cases in which I tried these injections they did no good, and Simpson found no benefit from injections of salicylate of soda and tannic acid. The bowel was not opened because the passage of the membranous stools ceased, and the autopsy did not reveal anything that operation could have removed.

Probably future experience will show, when the patient has tried all other means of relief without success, and when suffering is so great that life is a burden, that the best treatment is to open the colon on the right side, to allow the fæces to pass through the artificial anus for some months, and thus to give the diseased bowel complete rest. The artificial anus may then be closed. I believe the first published case in which this treatment was adopted was one sent to me by Dr. Walter Smith, which was brought before the Clinical Society in 1895 by Mr. Golding-Bird and myself (21). The patient was much benefited by the operation, but unfortunately she died (probably from the bursting of a pelvic abscess connected with old-standing pelvic trouble) five weeks after closure of the wound. Dr. Simpson (42) has published a case in which colotomy was done for membranous colitis in 1894 by Mr. Keith, and to him I believe belongs the credit of having been the first to perform the operation. The artificial anus was not closed for seven and a half months. Two years after, the patient was very well; only once or twice had she passed any muco-membranous matter. In February 1896, I saw with Dr. A. E. Taylor of Acton a lady who had been unsuccessfully treated for five years, and whose life was a perfect burden to her. Right colotomy was performed, the wound was left open nearly a year, and the patient improved immensely in all respects. Mayo Robson (39) has done an inguinal colotomy, and left the artificial anus open five months for a case of proctitis, in which ulcers could be felt from the anus. The patient was much relieved, and after closing the artificial anus she continued to do well. He is inclined to attribute much benefit to his having frequently washed out the bowel from the artificial to the natural anus; but although Mr. Golding-Bird and I did this we found that it brought away no membrane, so we discontinued it. Experience alone can show how long the artificial anus should remain open; probably the time will vary in different cases, but to be on the safe side it should be measured by months. I would lay great stress on doing a right colotomy rather than a left, for by means of it we are so much more likely to be well above the disease.

Secondary membranous colitis.—This condition seldom gives rise to any symptoms during life. Patients affected with it hardly ever pass membranes by stool. As dyspeptic membranous colitis is rarely fatal, the phrase membranous colitis, as used in the dead-house, nearly always refers to the secondary form.

i. Injury to the colon may be the cause of the disease, as in the case

of a little boy admitted under Mr. Howse in 1891. A cab-wheel pass over his abdomen and he died in three days. The mucous membrane of the colon was found deeply injected, and had a thin layer of granular lymph on it; and the muscular and serous coats were lacerated in several places. More often the injury is done by some corrosive substance, as in the case of a woman who swallowed three ounces of dilute sulphuric acid. At the post-mortem it was found that a large portion of the mucous membrane of the stomach was detached, and the colon and the lower part of the ileum were covered with false membrane. A woman who was in Guy's Hospital, nearly forty years ago, for a strangulated hernia, had taken much purgative medicine and had swallowed a quantity of duck shot. The hernia was easily reduced, but she died, and the whole of the colon and the last three feet of the ileum were in a state of membranous inflammation. Another woman swallowed a quantity of corrosive sublimate, and the whole colon was found in a condition of membranous colitis. The mercury was probably absorbed high up in the alimentary tract and excreted into the colon, which is certainly one channel of excretion for this metal. The possibility of this is shown by the case which Virchow brought before the Berlin Medical Society (46), in which severe membranous colitis was present as a result of mercurial poisoning following inunction and intravaginal injections of corrosive sublimate. Liebreich mentioned that he had produced the same condition in animals by subcutaneous injection.

ii. The septic variety of secondary membranous colitis is so neglected in text-books that I will briefly quote some cases from the post-mortem records at Guy's Hospital. 1893.—(*a*) A woman, æt. 68, suffered three or four days from a strangulated umbilical hernia of the transverse colon. Three or four inches of gangrenous gut were excised. In the cæcum and ascending colon there was extensive membranous colitis, where the mucous membrane was not covered with a thick white pellicle it was reddened. (*b*) A woman, aged 30, was suddenly and prematurely confined on July 24th; a dead fœtus and a quantity of blood were found in the bed. She had severe fever and abdominal pains, and died August 2nd. With the exception of the lower part of the descending colon and the sigmoid, the large intestine from the anus to the cæcum was covered with a greenish black membrane. The walls were thick and sodden. The kidneys weighed 17 ounces, and were in a state of acute non-suppurative inflammation. 1892.—(*c*) In a woman, æt. 38, the following events took place: premature labour, craniotomy, version, subsequent pyæmia, death in ten days. More than fifteen inches of colon near the sigmoid were covered with disseminated patches of membranous inflammation distributed upon healthy mucous membrane. The membrane was dark. General peritonitis and abscesses in liver and kidney were also present. (*d*) A man, aged 71, had gangrene of the foot; he died from exhaustion six days after admission. In the rectum and again above the sigmoid were irregular patches of membranous colitis. The membranes were gray and leathery in appearance. (*e*) In a woman, aged 27, a suppura

ovarian cyst ruptured into the peritoneum; she died in five days: there were patches of membranous exudation in the cæcum only. (*f*) A woman, aged 61, was admitted on February 2nd for pyuria and cystitis. On February 6th diarrhœa and hæmaturia set in, and she died exhausted on the 14th. Just within the anus and extending for three inches up in the rectum was a large area of brownish-gray coagulation necrosis, together with congestion and submucous hæmorrhages as high as the splenic flexure. The whole thickness of the bladder was necrosed and there were abscesses in the kidneys. 1891.—(*g*) A man, aged 39, first taken ill on March 26th, was admitted on April 2nd for cellulitis of the neck; this was incised and much foul pus escaped. Death, April 9th. At the autopsy necrosis of thyroid cartilage, probably due to acute suppurative perichondritis, was found. In the ascending colon the mucous membrane was reddened, and there were several whitish exudation patches with a red border.

It is curious to note that this batch of cases of septic membranous colitis occurred in the post-mortem room at Guy's Hospital during the three years 1891, 1892, and 1893. Looking back for many previous years I do not find any other cases. We have already seen that in 1886 there was a batch of cases of septic non-membranous colitis. Dr. Goodhart has pointed out how in some periods pyæmia is frequently associated with malignant endocarditis; and it appears also that at times it is liable to be associated with colitis either of the simple or membranous variety. It is not common in either case to meet with diarrhœa or other symptoms which point to implication of the colon; nor is it usual to find a general pyæmia, for in none of the membranous and in one only of the simple cases was there pus in the joints. In three of the seven membranous cases the kidneys were affected—two had abscesses, and one was in a state of non-suppurative inflammation; an interesting point as one of the simple cases had a peculiar condition of kidney. Five of the seven membranous and four of the five simple cases were in women; but perhaps these figures are to be explained by the liability of women to septic infection through their pelvic organs. In the report of one obstetric case it is noted that there were other cases of septicæmia in the ward about the same time. The membrane usually had a distinct line of demarcation, and there may be several membranous areas separated by healthy mucous membrane. Some of these cases appear to indicate that a considerable amount of membrane may form in seven or eight days.

iii. Wilks and Moxon mention that membranous colitis may be met with in Bright's disease; so the simple, membranous, or ulcerative forms may complicate this malady.

iv. Bristowe says that membranous colitis may occur with pneumonia. I have already given examples of the association of pneumonia with simple colitis.

v. A man, aged about 27, recently died under my care from coma due to rapid and severe diabetes. The mucous membrane of the first foot of the colon was acutely inflamed and had on it many patches of coagula-

tion necrosis. The inflamed area was sharply limited at one end by the ileo-cæcal valve, but the limitation at the other end was indistinct. There was also membranous inflammation of the lower end of the œsophagus, and the stomach was much reddened.

vi. Membranous colitis may be associated with cancer. It is very rare, but this year a woman died under the care of Dr. Pye-Smith who during life had passed much membrane. At the autopsy an annular stricture at the upper part of the sigmoid was found with a few shreds of adherent membrane.

Many authors describe diphtheritic colitis, but, as we cannot accept any membrane as genuinely diphtheritic until the Klebs-Löffler bacillus has been demonstrated in it, the existence of diphtheritic colitis is at present an open question. It is stated that membranous colitis may be found in persons dying from various other specific fevers, or tubercle. Fat necrosis of the peritoneum has been seen in cases of membranous colitis. It is uncertain whether the bacillus coli communis has anything to do with the production of this disease.

III. Ulcerative colitis.—The colon is frequently ulcerated as a result of typhoid fever, dysentery, tuberculosis, or malignant disease; but from time to time we meet with cases in which this part of the bowel is extensively ulcerated quite apart from any of these diseases. Observation at the bedside has shown that nearly all patients in whom this independent ulceration is found after death have, during life, presented such a grouping of symptoms as to enable us to predict that the colon will be found ulcerated. To this disease, with its characteristic symptoms and characteristic morbid anatomy, the name simple ulcerative colitis, or more shortly ulcerative colitis, is applied. It is a bad name, because the colon is ulcerated in other diseases; but it is so generally used that much confusion would be caused by an alteration of it at present.

Etiology.—The cause of this disease is unknown; but some day it may be found to be due to a micro-organism acting locally on the colon; and, possibly, different varieties of it are caused by different but closely allied micro-organisms. Neither occupation, place of residence, nor any previous illnesses, except Bright's disease, have, so far as we know, any relationship to it, but one of my patients said she had always been liable to diarrhœa since an attack of typhoid fever two years before. Statistics would seem at first sight to show that it has been more commonly met with of late years than formerly; but this is probably due to the fact that we have only recently learnt to recognise it.

Age and Sex.—The average age of eleven cases, of which I published an account (50), was 40 years. The youngest was 17, the eldest 59. In 1893 (*Guy's Hospital Gazette*) my house physician, Dr. F. J. Colman, collected 17 additional cases, and found the youngest was 21, and the eldest 54. Ulcerative colitis, therefore, is probably not a disease of childhood, nor of old age; and this conclusion is supported by Dr. Tooth's recent paper. If we may judge from the cases we have had at Guy's, it is not appreciably commoner in one sex than in the other; for out of 28 cases

13 were women and 15 were men: but more female than male cases have been shown at the Pathological Society.

Symptoms.—It is nearly always the state of the bowels which seriously directs a patient's attention to his illness, and the first symptom which he remembers is usually abdominal pain, generally griping, sometimes very sharp and severe, and often sudden in its onset; in one case the patient shrieked under it. Soon it disappears only to reappear later; the duration of these painful attacks varies from a few minutes to many hours, and their alternate appearance and disappearance is very characteristic. The pain is always referred to the front of the abdomen; but its exact position varies in different cases, and also in different attacks in the same patient. Occasionally there is also pain in the back and loins; and once I have known a patient complain of such severe pain in the front and sides of the chest that she was incorrectly thought to have pleurisy as well as ulcerative colitis. In acute cases the first attack may be excruciatingly severe; but often it does not cause much suffering, the intensity of the pain increases, however, in each succeeding attack. In the interval between the attacks the patient is usually quite free, or he may complain of a dull pain in the abdomen. It should be mentioned that the pain bears no relationship to the ingestion of food, but is commonly worse when the bowels are open. The patient lies quiet on his back during an attack; but if perforation has occurred the legs may be drawn up. The mere presence of ulcers is insufficient to explain the pain, for it is commonly absent in typhoid fever; it is probably due to some peculiarly irritating ingredient of the contents of the intestines which stimulates the nerves exposed on the floor of the ulcer, and thus sets up irregular peristaltic contractions. In the majority of cases there is no abdominal tenderness; when this is present it is rarely intense, and most often it is especially marked over some part of the colon.

The presence of severe diarrhœa—sometimes interrupted by short periods of constipation—is almost of equal symptomatic importance to the pain; it is often the first symptom noticed, and these two symptoms are never absent throughout the whole of a case. The frequency with which the bowels are open commonly varies between 2 and 11 or 12 times in the twenty-four hours; but in one of Dr. Sharkey's patients the bowels were open from 10 to 24 times in the twenty-four hours. A man aged 22, who died of ulcerative colitis in Guy's Hospital, had his bowels open the following number of times on each of the 14 days before he died, 6, 5, 2, 6, 7, 4, 5, 5, 8, 6, 6, 6, 5, 11. Although the act of defæcation is often accompanied by abdominal pain, there is rarely the intense tenesmus characteristic of dysentery; nor have I heard of a patient complaining of that almost constant desire to go to stool which is so frequent in a severe dysenteric attack. If the case is mild an occasional solid motion may be passed; but the evacuations are nearly always fluid, dark, foul-smelling, and of a consistency varying between that of slime and water. So far as our present knowledge goes, they never resemble ordinary dysenteric or choleraic stools; indeed, they are so

unlike them that probably the distinction is of considerable diagnos value. Blood is very commonly present, especially after a period of co stipation, sometimes as but a trace, sometimes in large quantities; the latter case the presence of much clot—when a patient describes t motion as being like red-currant jelly—forms an exception to the sta ment that the motions are fluid; but usually the blood is liquid, and then bright red, showing that the bleeding has been recent. Commo it is passed with the motion, although not intimately mixed with it; a if the latter contain any solid particles small clots of blood will adhere them. Occasionally it distinctly follows the motion, and rarely it p cedes it. These differences probably depend upon the anatomical relat of the bleeding ulcer to the motion which is evacuated. The clots n be flat and smooth on one surface and shreddy on the other, suggest that they have been recently detached from the floor of an ulcer. I few instances the patient was unaware that he was ill until he detec blood in his motions.

Although after constipation a motion consisting of little but s fæces may be passed, yet when diarrhœa is present very little fa matter is seen in the evacuations, and what there is consists of a small lumps scattered about in the fluid. In some cases a little mu has been observed, but it is never present in large quantities, nor in form of little transparent pellets, as in cases of follicular ulcerat Often shreddy masses looking very like sloughs are seen: they much in colour and size; under the microscope they are generally st tureless, although occasionally leucocytes may be noticed, and there be indications of epithelial cells; probably their structure has l destroyed by the action of the contents of the large intestine. In on two cases a little pus is said to have been present in the evacuations, this is certainly exceptional; enough pus to be easily recognisable is n seen. In one case, in Guy's Hospital, the amœba coli was found. the other symptoms about to be described are superadded to the and diarrhœa.

The most frequent of these, and one of the earliest, is vomi Often when patients are admitted into hospital they say they vomited incessantly, or have brought up everything they have ta but usually under rest in bed and careful dietary the vomiting bec much less. Sometimes it is an unimportant feature in a case; patients indeed never suffer from it. I have known the vomi contain blood in a case in which the ulceration was confined to the l intestine; the blood came, no doubt, from the rupture of some s vessel in the stomach, throat, or gullet; a matter of no surpris these patients frequently have granular kidneys. Neither the vomi the constipation, nor the diarrhœa stand in any proportion to the ar ulceration, but, like the pain, are to be regarded as due to the irritatic nerves exposed on the floor of the ulcer. The loss of fluid by vomiting diarrhœa is probably the cause of the thirst, which may be a great tro Nausea may be complained of, either without vomiting or in associa

with it. The tongue is usually covered with a dirty white fur; but as the disease progresses it becomes red and dry with a brown fur. If he be seriously ill the patient has the drawn face characteristic of abdominal disease, and occasionally there is considerable sweating. The abdomen is commonly distended, and the peristaltic movements of the intestine may be visible. A rectal examination should never be omitted, because, as in a patient I saw recently, it is sometimes possible to feel the ulceration; in one case, which occurred before ulcerative colitis was a recognised malady, the surgeon, feeling an ulcer, concluded that the patient had malignant disease of the rectum or sigmoid flexure, and performed left lumbar colotomy. At the post-mortem examination on a patient under my care in 1893, one large and several small ulcers, of sufficient age for one of them to have extended through the internal sphincter into the perirectal connective tissue, were found just inside the anus. During life the diagnosis of ulcerative colitis was obvious, but I had examined the patient's rectum three weeks before death, when the whole of the mucous membrane which could be reached by the finger was found intact. The ulcerative process may, therefore, extend rapidly.

The general symptoms are such as would naturally be expected. The patient gradually becomes wasted, anæmic, and excessively weak, so that he lies in bed looking bloodless, sallow and extremely ill. No doubt these symptoms are due, to a considerable extent if not entirely, to the diarrhœa and loss of blood. The pulse is feeble, and hæmic murmurs may be heard. Irregular pyrexia is often present, the temperature ranging between 100° and 102°; but towards the end of the illness, if the patient be very collapsed and exhausted, it may be subnormal. The highest temperature I have seen was in a girl who was in the Hospital from October 24 till her death on November 12; her temperature was always between 102° and 103°, often over 103°, and twice it reached 104°. A girl in the Hospital in 1890, under Dr. Goodhart, had a temperature which ranged between 102° and 104°; and Dr. Sharkey gives a case in which 104·6° was attained. Towards the end of the patient's life there may be muttering delirium and other symptoms of the typhoid state. The cause of death is usually exhaustion, and in some cases it is distinctly accelerated by hæmorrhage. Occasionally perforation of the floor of an ulcer with consequent peritonitis is responsible for the result; and in one patient no perforation could be found although acute peritonitis was present: as the intestinal inflammation was very severe, it appeared probable that the peritonitis was due to direct extension of the inflammatory process through the walls of the gut. It is noteworthy that although, as will be shown directly, ulcerative colitis is frequently associated with granular kidneys, sufferers from it do not often show signs of uræmia.

Prognosis.—Although patients who come into the Hospital, and who are thought to be suffering from ulcerative colitis, are occasionally discharged cured, still the prognosis is very grave—so grave, indeed, that it is quite likely that most of these patients were not suffering from ulcerative colitis at all; this diagnosis has sometimes been confidently made

when post-mortem examination has shown the bowel to be only reddened. On the other hand, even fatal cases occasionally show an attempt at repair of the ulcerated surface. Therefore, all we can say is, that while certainly the mortality is very high it is not yet proved that the disease cannot end in recovery. The shortest case I know was in a man who died collapsed soon after admission to the Hospital. He said that three days previously he had been suddenly taken with severe diarrhœa and abdominal pain. In the colon were several large acute ulcers. At first sight it might be thought that the acuteness of this case was due to the fact that there were also eight or ten similar ulcers in the small intestine from the duodenum downwards; but another case, in which ulcers were found high up in the small intestine, does not support this view. Occasionally patients state that they have been liable to diarrhœa for a long time—in one case as long as two years—but this is quite exceptional; the patient is usually dead within eight weeks from the commencement of the illness. The symptoms which especially indicate a speedy termination are great tympanites, much loss of blood, very profuse diarrhœa, and occasionally a high temperature.

Diagnosis.—Although, no doubt, confusion might arise between ulcerative colitis and dysentery, in practice they are not often confounded, for both diseases are rare in England. I have already said that the violent tenesmus, the burning pain at the anus, the constant desire to go to stool, and the scanty meat-washing evacuations which are characteristic of dysentery help to separate it from ulcerative colitis. Then, too, there is the lack of a history of residence abroad and of dysentery contracted there. If it be ultimately established that dysentery is due to a particular variety of amœba, the presence of this in the motions may help in diagnosis; but it is quite possible that as the morbid anatomy of ulcerative colitis is much the same as that of dysentery, the former disease may be due to another variety of amœba. It is highly probable that a point of practical distinction between the two diseases may be that ulcerative colitis is hardly ever as acute as an acute case of dysentery; while on the other hand it is never so chronic as are most of the cases of chronic dysentery that we see in England.

In actual practice the difficulty of diagnosis is usually to separate ulcerative colitis from malignant disease of the large intestine, from intestinal obstruction, and from some form of primary anæmia. The differential diagnosis from the first of these will readily occur to the reader. A mistake is usually due rather to faulty examination of the patient than any real difficulty. Fortunately the form of intestinal obstruction most likely to be mistaken for ulcerative colitis is intussusception, which, however, is very rare at the age at which ulcerative colitis is most common: still the importance of diagnosis is so great that whenever a patient comes before us with what we consider to be either intestinal obstruction or ulcerative colitis we must never forget the possibility of error. I have known the severity of the anæmia in a case of ulcerative colitis suggest some primary blood condition, but after a few days of careful observation the difficulty

cleared up. I have never heard of typhoid fever simulating ulcerative colitis; but one patient, who also had severe bronchitis, was at first thought to be suffering from tuberculosis; the pulmonary signs, however, remained stationary, and a correct diagnosis was soon formed. Once I saw ulcerative colitis overlooked in a patient who had a large abscess in his liver; the diarrhœa from which he suffered was supposed to be due to the leakage of the abscess into the intestine. However, he was moribund when seen. It is worth while to repeat that as the ulceration often extends low down in the rectum, a rectal examination should never be omitted; if necessary, an anæsthetic should be given and a speculum used. It may be well also to warn the physician against a diagnosis of syphilis in such a case; in syphilis the anus is always implicated.

Morbid anatomy.—Any part of the large intestines may be affected, and generally the ulceration has no special distribution; but in one case it is said to have been most marked along the mesenteric attachment of the bowel: in another it appeared to follow the longitudinal muscular bands. A typical case of my own, in addition to ulceration of the colon, showed ulcers in the vermiform appendix. In an extreme case the muscular coat is exposed, the floor of the ulcers can be distinctly seen to consist of the bundles of muscular fibres, and the ulceration is so extensive that only islets of mucous membrane are left here and there: often these are considerably swollen, and consequently they look taller than they otherwise would do; and frequently they are more or less stalked because of the ulceration which undermines them. The result of this is that a careless observer takes the islets of mucous membrane for polypoid growths, and the exposed muscular coat for the natural level of the colon. The colon may be much dilated, and where it is not ulcerated the muscular wall is sometimes hypertrophied. The peritoneal surface over the ulcer is usually normal; but it may be much congested, and if the floor of the ulcer is sloughing there may be a little local peritonitis around it. The vessels of the mucous membrane are dilated, and sometimes the membrane is black, as though from long-standing congestion. The number of ulcers varies from one or two to several dozens; but while ulcers of recent date are often more or less circular, and vary in size from a pea to a five-shilling piece, by the time death occurs their shape is usually very irregular; and occasionally, like all chronic intestinal ulcers, they show a tendency to become transverse. When numerous they run one into the other, and produce a series of ulcerated surfaces so irregular in shape, that the comparison made by MM. Bertrand and Fontan to the irregular pieces of a child's geographical puzzle is very apt. Generally at the time of the autopsy no sloughs are to be seen; but in a few cases one or two ulcers have shown a ragged surface, indicating that a slough has been recently detached, and if the patient has died early in the disease they may be seen adherent to the ulcers. It is extremely rare to see any attempt at repair; in one case the account of the post-mortem examination states that in both the large and small intestine there were several pigmented spots of old ulcers, but, so

far as I know, there is never any evidence of constriction of the bowel. Sometimes the ulceration extends right through the muscular and serous coats, and perforation results; this may happen in any part of the cæcum, colon, or rectum. In one case the floors of the ulcers were so thin that they bulged, forming little pouches. Rarely, as in cases recorded by Dr. Ormerod and Dr. Lazarus Barlow, the perforations are very numerous. These numerous perforations, which are also seen in some cases of typhoid fever, are difficult to explain; for as the patients, if we may judge by their symptoms, do not long survive the first perforation, there seems hardly time for the others to appear. Perhaps the explanation lies in the fact that the peritonitis set up by the first perforation so softens the floor of other ulcers that they rapidly give way. All the ulcers may be in an advanced condition, but if the patient die early in the course of the disease the mucous membrane of the colon is found to show a highly vascular, soft, red surface covered with many recent ulcers. I have recently seen a case in which the whole colon was in this state; there must have been more than a hundred superficial ulcers varying in size from a split pea to a threepenny piece. When the disease is in this early stage the wall of the bowel is so softened that it is sometimes described as rotten. It may be possible in the same case to see all gradations between the early and late stages of the process; and even when the ulcers are of a considerable size and depth they may be still surrounded by a very red halo of congestion. The disease seems, therefore, to go through just the same stages as an ulcerative process in other parts of the body. There is never any satisfactory naked eye or histological evidence that the process began by ulceration of the solitary follicles, although in a few specimens the follicles may be seen to be ulcerated, and in one case many of them contained a bead of pus; so unimportant, however, is follicular ulceration in ulcerative colitis that it was seen in none of the eleven cases I published in 1888 (50). The difference between the two conditions will be given more fully when we come to speak of follicular ulceration. The mesenteric vessels are never mentioned as thrombosed; and the account of the post-mortem examination rarely states that the mesenteric glands are enlarged. It is clear from this description that it is often quite impossible to tell from looking at the bowel whether the patient died of dysentery or ulcerative colitis; but I have already pointed out the many clinical reasons for separating the two diseases.

In some cases the small intestine is affected as well as the colon, but this is rare, and the process is much more severe and apparently of longer standing in the colon. In one case there was also a small ulcer in the stomach.

The liver is often fatty.

Histologically the process is one of acute inflammation. When a patient dies early in the disease the hyperæmia is, as just mentioned, sufficiently marked to cause considerable redness of the mucous membrane, which becomes soft and swollen, and may contain small hæmorrhages. In the next stage there is a considerable accumulation of small cells in

the submucous layer. This severe inflammation leads to necrosis of portions of the mucous membrane, which is got rid of either as small particles or as sloughs. In this way ulcers of various depths are produced, leaving the swollen islets of mucous and submucous membrane. Commonly the muscular coat is not much affected, but some increase of leucocytes may be seen in it. Frequently the only change in Lieberkuhn's crypts is that the epithelium is cloudy and swollen; where there is ulceration they are cut off flush with the ulcer, unless it be so deep that they are destroyed. Occasionally the upper part of the follicle gets blocked, with the result that the lower part of the gland is much distended.

Associated diseases.—About half the patients who die of ulcerative colitis show no disease in any organs of the body except the intestines, and, if perforation has taken place, the peritoneum. The coincident diseases that in any way suggest a special association are Bright's disease, gout, and suppuration of the liver. The association of chronic interstitial nephritis with ulceration of the colon has long been known. Case 23 of Bright's "Tabular View of the Morbid Appearances occurring in 100 Cases in connection with Albuminous Urine" (5) is that of a woman aged 40, in whom the kidneys were "hard, rough, and lobulated," and the large intestine was "ulcerated throughout." Mr. H. B. Robinson has recently recorded a case in the *Pathological Society's Transactions* which shows the relationship very well, and another has been described by Dr. Bannatyne (3). Among the twenty-three cases of which I have notes of the condition of the kidney, chronic interstitial nephritis was present in six, and in at least four of these the renal change was advanced. One of the six patients was also suffering from lead poisoning, and two of the seventeen whose kidneys were healthy had urate of soda in their joints.

The association of abscesses in the liver with ulcerative colitis is rare, and requires a little consideration. It is well known that small multiple pyæmic abscesses, accompanied by rigors and the usual symptoms of pyæmia, may occur when there is any source of infection in the periphery of the portal vein—such as an ulcer of the stomach or intestines, an anal fistula, an operation for piles, ulceration of the gall-bladder, abscess in the spleen, disease of the pancreas, appendicitis, or disease of the female pelvic viscera. But when we consider how common many of these conditions are, it is remarkable that portal pyæmia is so rare. In thirty consecutive cases of ulcerative colitis, which have either been under my care or of which I have read the description of the autopsy, pyæmic abscesses of the liver were only found once.

There is very strong evidence that some forms at least of dysentery are due to an amœba, and that this microbe can be taken up by the portal vein and produce in the liver the large solitary tropical abscess which is so well known [*vide* art. "Amœbic Abscess of Liver," vol. iv.] This usually occurs in those who have dysenteric ulceration of the intestine, and may occasionally be associated with multiple pyæmic hepatic abscesses due to the intestinal ulceration; or it may occur without any dysenteric ulceration; and as in these cases the amœba has

been found in the pus of the hepatic abscesses it appears that this microb like the scolices of the tænia echinococcus, can without any intestin ulceration gain access to the radicles of the portal vein, and, witho causing any intestinal ulceration, set up a suppurating cyst. We may no in passing, the curious fact that although the amœba and scolices m swarm in the intestine the abscesses they set up in the liver are sing These facts have a parallel in ulcerative colitis. Instances of la abscesses of the liver in association with ulcerative colitis have be recorded by Fagge (18) and Dickinson (11), and the recent records post-mortem examinations at Guy's Hospital contain another instance i woman aged 30. Two years ago I had under my care a case of ulcerati colitis, in which the sequence of events was difficult to interpret; but was very probable that the patient, like the cases of dysentery just m tioned, had both the large solitary form and the multiple pyæmic vari of abscess in his liver (53). Lastly, it may be that those cases of solit abscess of the liver occasionally found in persons who have never be abroad, and who have received no injury, are due to the fact that t micro-organism of ulcerative colitis has, like the amœba of dysente reached the liver without causing any intestinal ulceration.

Treatment.—Unfortunately this avails but little. The patient m remain in bed and be kept warm. If the pain be very severe l abdominal fomentations are comforting. The diet should consist of mi custard, jelly, blancmange, and such like articles of food; vegetables a fruits should be avoided, and beef tea will probably increase the diarrh Three or four ounces of brandy every twenty-four hours are generally use Bearing in mind the abdominal pain, and that the diarrhœa is oft excessive, opium is probably the best drug to prescribe. In a severe c the patient should be kept well under its influence; it matters lit what preparation is given. As a rule a grain every four hours m be ordered to begin with, and if this does not suffice the dose may gradually increased; sometimes a starch and opium enema is m efficacious in checking the diarrhœa than opium by the mouth. course, if the patient be suffering from interstitial nephritis it w be necessary to use great caution in prescribing opium; in any c it is doubtful whether it is wise to push it to such an extent as cause actual constipation. Half or three-quarters of a pint of a satura solution of boracic acid at about 100° F. may twice a day be let to r slowly into the bowel through a long rectal tube introduced as far possible, while the hips are well raised. Considering, however, extremely intractable nature of the disease, I am disposed to think t it may be justifiable, in some cases, to make an artificial anus just ab the cæcum, to let the fæces be discharged through it, and then to syri the whole of the large intestine from the artificial anus to the natu anus with boracic solution twice a day. The artificial anus could closed later as in membranous colitis.

To relieve the vomiting a drop of tincture of iodine in half an ounc water, or three drops of dilute hydrocyanic acid in an ounce of an ef

vescing mixture, may be given every hour. If these fail, trial may be made of ten grains of each of bicarbonate of sodium and carbonate of bismuth suspended in an ounce of water with sufficient mucilage. If the rectal hæmorrhage is dangerous, an ounce liquor ferri perchloridi diluted with three ounces of water should be injected per rectum as far up the colon as possible.

IV. Follicular ulceration.—This condition is far more common in the large intestine than in the small. It begins with an accumulation of small round cells in the solitary follicles, which therefore swell. Soon the epithelial covering dies and ruptures, and then there follows disintegration of the follicles, the result being many small round ulcers, beginning as a minute depression in the centre of the follicle, and having sharply cut edges, perhaps a little overhanging, but never bevelled. These ulcers do not extend deeply, although they may extend laterally a little beyond the follicle; for the process of disintegration usually advances a little way in the submucous coat round the follicle, and the ulcer then becomes somewhat flask-shaped. In a well-marked example the gut is quite honeycombed by these circular ulcers, which vary in size from a hemp seed to a large pea. I have not met with any case in which there was cicatrisation; in fact, none of the cases in our autopsical reports is mentioned as healing; nor is perforation ever recorded, the floor of the ulcer being always the muscular coat. Occasionally ulcers are described as running together to form an irregular ulcerated surface, but this is not common. The mucous membrane between them is not usually affected, except that in their immediate vicinity there is a considerable collection of small cells. Sometimes at the post-mortem examination a bead of pus will be found in some solitary follicles, and if the patient had lived this minute abscess would have burst, and a follicular ulcer would have resulted; but it is by no means certain that all follicular ulcers are preceded by suppuration.

The patients in whom follicular ulceration is found have always died of some other disease, often of the gastro-intestinal tract; thus I find that of ten adults one had dysentery, one cancer of the rectum, one membranous colitis, one typhoid fever, and one a femoral hernia. Of the other five, three were the subjects of tubercle, but none of them happened to have tubercular ulceration of the intestine; one died of multiple sarcoma, and the fifth of a retro-peritoneal malignant growth. Ten instances are too few for the deduction of averages; but in order that they may be added to future cases I may state that of the ten adults seven were males; and that the ages varied from 17 to 54. Follicular ulceration of the colon occurs about once in every 500 post-mortems made at Guy's Hospital. It is never diagnosed during life, for the symptoms of the fatal disease quite overshadow any that might be attributed to the ulceration. Occasionally it is seen in the small intestine, and there it does not differ from the ulceration as it occurs in the colon. Follicular ulcers are rarely found in cases of ulcerative colitis; when they are present they should be regarded as a complication, for there is no reason to believe that ulcerative colitis begins particularly in the follicles.

Inflammation of the solitary lymph nodules of the colon, going on to follicular ulceration, is proportionately more frequent in children than in adults. Thus Holt met with follicular ulcers in the intestine in twenty out of seventy fatal cases of infantile non-tubercular diarrhœa. They were never seen in cases that had lasted less than a week, and the proportion of follicular ulceration was highest in those cases which had lasted more than ten weeks. He found that cases with considerable follicular ulceration ran a slower course than other varieties of diarrhœa, and that marked pyrexia and blood in the stools were exceptional. In two of the twenty cases the ulcers were in the small intestine only; in fifteen, in the colon only; and in three, in the colon and small intestine. When in the small intestine they were always near the cæcum. The commonest seat for the ulcers was the descending colon and sigmoid, and in this region they were most numerous, largest, and deepest. In children as in adults it is very rare to find any evidence of healing in follicular ulcers. Softening in the centre of a follicle often occurs as an early post-mortem change in the intestines of children; a depression forms, and the condition may be mistaken for early follicular ulceration.

It is common in persons dying of various diseases, especially in the colon, to find the follicles enlarged, swollen, and prominent without any ulceration. I have noticed that they are especially well marked in fatal cases of exophthalmic goitre.

In children the solitary follicles of the intestine in the normal state are hardly visible to the naked eye; but in the large intestine they are often seen projecting above the surface; and they may have a black dot in their centre, when the intestine presents the well-known shaven beard appearance. This is especially common in children who have died of diarrhœa.

V. Vascular ulceration. — This is clearly of two kinds, *venous* and *arterial*.

With regard to the *venous*, I cannot do better than quote from Wilks and Moxon, who say: "Ulceration of the large intestine is one of the most common post-mortem appearances we meet with; in persons long ill with various visceral complaints we often find large ulcers in various parts of the large bowel, but more especially in the cæcum, ascending colon and sigmoid flexure. These are mostly of a chronic character with raised indurated edges; some spreading, while others are healing. They probably have the same pathology as many ulcers on the leg connected with retardation of the venous circulation. These ulcers have their length in the direction of the transverse course of the bowel, passing around its circumference. In cases of phthisis and tubercular disease it is not uncommon to find such ulcers, and not apparently having a tubercular character."

As the circulation through the portal vein is probably somewhat impaired in cases of cirrhosis of the liver, it is not surprising to find venous ulcers of the colon in association with this disease. An instance occurred to me last year, in a man who had died of cirrhosis of the liver, and in whom

an ulcer was found in the colon 3 feet 6 inches above the sigmoid flexure. I find that at Guy's Hospital these venous ulcers, showing no trace of growth or tubercle, are also found chiefly in persons who have died of phthisis or malignant disease. Thrombosis of the mesenteric veins affects the small intestine as well as the large; but in either it will cause much reddish lividity and swelling of the mucous membrane. This may go on to ulceration.

There are at least three varieties of ulceration of the colon produced through the *arterial* system.

The mesenteric vessels may be blocked by emboli, and thus ulceration may complicate malignant endocarditis. In a well-marked case the embolism leads to gangrene of the bowel, hæmorrhage into its swollen coats, and dilatation of the veins; and the ulceration which follows may lead to perforation. Although peritonitis usually supervenes even if no perforation has taken place, I was once much struck with the fact that while the interior of the bowel was gangrenous there was no local peritonitis. The main symptoms are intense abdominal pain and tenderness, and, if the patient live long enough, there may be tympanites and foul stools containing blood; but it is remarkable that severe results of obstruction of the mesenteric vessels may be found post-mortem, although no symptoms have been present during life. This variety of ulceration has been produced by artificial embolism.

In the second variety ulceration of the colon may be caused by thrombosis of the mesenteric artery or of one of its branches. The following are good instances of this; the first was a remarkable case under Mr. Howse (29):—The patient, a woman aged 49, had gangrene of one of her legs. At the post-mortem examination the corresponding femoral artery, the aorta, and the superior mesenteric artery were thrombosed. There was peritonitis, a communication between the small intestine and ascending colon, and another between the small intestine and descending colon. The descending colon contained numerous small pigmented ulcers; in the cæcum there was an extensive ulcer, and in the small intestine many sloughy ulcers were found. A woman, aged 66, who was under Dr. Newton Pitt, died in 1891: she had mitral stenosis and thrombosis, which completely occluded the inferior mesenteric artery. The colon and last five feet of the ileum were intensely engorged and ecchymosed. This change would probably have gone on to ulceration. Some authors mention that the pressure of tumours on the mesenteric arteries may lead to ulceration of the intestine. In the following case this had happened, in the small intestine. A woman, aged 60, had a large lymphomatous mass in her mesentery. The corresponding part of the small intestine was in parts deeply stained with bile and sloughing; in other parts the mucous membrane had sloughed, leaving an ulcer; and at one spot there was an ulcer three inches long.

The third variety of arterial ulceration is that which depends upon imperfect circulation through the mesenteric arteries, owing to atheroma of the aorta. The following case is a good instance of this:—

A man, aged 66, was admitted in 1880 under Mr. Bryant for gangr of the legs. Most of the vessels of the body were extremely atheromato and the athéroma of the aorta had much diminished the orifices of : mesenteric vessels. The colon, which had in some parts a worm-ea appearance, was extensively ulcerated.

VI. **Hæmorrhagic ulceration.**—In some diseases submucous extra sations of blood take place into the colon, and these, no doubt, may br down and lead to ulceration. Hæmorrhagic ulcers are usually sm shallow, rounded, and discrete, although occasionally two or three r run together; submucous hæmorrhages may commonly be seen in t neighbourhood.

It is well known that persons suffering from chronic Bright's dis are liable to hæmorrhages in any part of the body; and it is also kn to those who make many post-mortem examinations that these pati occasionally present ulcers in the intestines, or even in the stomach. W and Moxon drew attention to the fact twenty years ago. It may as Dr. Dickinson suggests (12), that submucous hæmorrhage is one ca for the ulceration. He gives eight cases in which submucous hæ rhages in the intestine were found in chronic Bright's disease, and liability of these patients to hæmorrhages was demonstrated by the that they were present in the retinæ of all those whose eyes w examined. Every one must allow that submucous hæmorrhages ma associated with ulcers in the colon, for it is proved by Dr. Dickinson's other cases. For instance, in 1888 a man who had been in G Hospital died under the care of Dr. Taylor; the kidneys were granu there were numerous superficial ulcers and submucous hæmorrhages i the cæcum and in the ascending colon. But after hearing Dr. Dickins paper, I could not help feeling that, although some intestinal ulcers fo in the subjects of Bright's disease are very probably hæmorrhagic, ye could not be thus explained; for it must be remembered that, as W and Moxon have taught for many years, enteritis and colitis are not common complications of this disorder, and we might fairly expect sometimes before death they would proceed to ulceration. Then ag ulcerative colitis is often associated with Bright's disease, and in a se case the swelling of the mucous membrane and the whole intensity of process, which may lead to the denudation of several feet of muc membrane, shows that the ulceration cannot have originated in submuc hæmorrhages. They may be present near the ulcers, it is true, but su they ought sometimes to be regarded more as evidence of the intensit the inflammation than as the cause of the ulceration.

The view here expressed that hæmorrhage, although a cause, is from being the sole cause of intestinal ulceration in Bright's disease, been recently supported by Dr. Bannatyne (3), who has reported two ca In one it is probable that the ulcers were hæmorrhagic, but the other an unmistakable example of ulcerative colitis.

Submucous hæmorrhages, leading to ulceration, may occasionally o in other general diseases—such, for instance, as purpura, scurvy,

severe forms of anæmia. In a case of Addison's disease under Dr. Carrington there were some small intestinal ulcers, and even the intestinal mucous membrane showed the pigmentation characteristic of the disease.

VII. Trophic ulceration.—Dr. Acland and Mr. Targett have both raised the question whether disease of the central nervous system can cause ulceration of the colon. Dr. Acland's first case (1) was a man aged 37, who died of syphilitic myelitis opposite to the fourth dorsal vertebra. There was some ascending degeneration in the posterior columns, but little or no descending degeneration in the lateral columns. The intestines were enormously distended, and there was early purulent peritonitis. "Scattered at intervals over the mucous surface of the small intestine were patches varying in size from $\frac{1}{8}$ to $1\frac{1}{2}$ inch, which were dark, almost black, and surrounded by injected vessels." There was a large patch just above the cæcum and one below it. The second case was also one of syphilitic disease of the cord. The colon and stomach were greatly distended. The former had scattered through it "small ulcers, usually in groups of four and five." "They were circular, one-eighth of an inch in diameter, and perfectly clean cut." On the lesser curvature of the stomach was a similar group of ulcers.

Mr. Targett's case (44) was a man aged 50, who died on the fifth day after fracture of the fourth and fifth lumbar vertebræ. The second day after the accident persistent diarrhœa set in, and it continued till death. There was no naked eye damage to the cord or cauda equina. The mucous membrane of the whole of the large intestine showed many superficial oval ulcers varying from $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter. There was a slough on the floor of many of these ulcers.

In the Guy's Hospital Reports (25) I recorded the case of a woman who had double descending lateral sclerosis, secondary to cerebral disease. Four days before death very severe diarrhœa set in. The colon showed intense colitis, the mucous membrane being rough, granular, and ecchymosed.

There is a specimen in the Guy's Hospital Museum showing enteritis of the lower part of ileum. The inflamed part is thickened and contracted, the mucous membrane is covered with granular lymph. All the coats of the bowel are infiltrated with small round cells. The patient was a woman aged 35, who was admitted under Dr. Pitt for hæmatemesis and severe anæmia. At the autopsy the left lobe of the cerebellum was softened, there was a large chronic ulcer in the stomach, and a few patches of superficial ulceration in the colon. There is also another specimen showing numerous small round ulcers which occupied the whole of the colon. In some the muscular coat is exposed; in others a thin pellicle covers the floor. This specimen was taken from a man, aged 51, who was admitted under Mr. Lane for fractured spine in the lumbar region with complete paraplegia. He died fourteen days after the accident, having had diarrhœa for some days before death. In another case of fractured spine in the lower dorsal and lumbar region, also under the care of Mr. Lane, the patient lived for two months, and post-mortem a

mild degree of colitis was found and one superficial ulcer in the colon. Blood was passed during life. A patient under the care of Dr. F. Taylor, who during life showed symptoms of locomotor ataxy and ulcerative colitis, recently died in Guy's Hospital. The spinal cord showed the changes characteristic of locomotor ataxy, and the intestine those of ulcerative colitis. Recently I have seen a patient with undoubted locomotor ataxy and all the symptoms of ulcerative colitis.

It would be unwise to draw any conclusions from so few cases, but these cases should direct our attention to the state of the colon or the small intestines in diseases of the nervous system. Seeing the ease with which bedsores and cystitis occur on lesions of the spinal cord, it is hardly surprising that ulcers are occasionally found in the bowel.

Dr. Cowan (10) has called attention to the frequent occurrence of enteritis, colitis, and intestinal ulceration in the insane, and is inclined to regard the intestinal changes as, in some cases, secondary to the nervous disease. But according to Eurich (16) the postulate is not at all proved; he lays great stress on the general lowered vitality of lunatics, rendering them an easy prey to disease of all sorts. Most of his cases in which the condition of the colon was not due to some recognised disease, either had gangrene of the lungs, and so might have had septicæmia, or they were filth-eaters.

VIII. Dilatation of the colon.—Cases in which the large intestine is dilated may be divided into four groups: (i.) The first contains those in which the distension is entirely gaseous, is not due to any obstruction, and is only one symptom of some other illness. It is often seen in association with peritonitis and typhoid fever, and it may occur with almost any severe illness. Usually there is at the same time some distension of the small intestine. When extreme, it is not only distressing to the patient, but it is of great importance, for it adds considerably to the danger of the original disease; it greatly hampers the movements of the heart and lungs, thus causes palpitation and difficulty of breathing, and, if it occur in association with heart disease, is particularly dangerous. In an extreme case the abdomen is very distended and tense; it hardly moves on respiration; it is hyper-resonant on percussion; the tympanitic note extends well into the flanks; the liver is pushed up so that the hepatic dulness does not reach to the lower margin of the ribs, and the splenic dulness is obliterated. If the distension or tympanites, as it is usually called, affect one part of the intestine much more than another, the shape of the distended part may be visible. The patients are nearly always constipated; they do not usually pass much flatus by the rectum, nor by the stomach; but they complain of borborygmi, which may be audible to bystanders (*vide* case, p. 509, Pat. d. of Heart Disease).

The gas in the intestine, according to Bertin, consists of carbonic acid and hydrogen, and Hoffmann (27), quoting from Planer and Ruge, says that marsh gas is also frequently present. At the post-mortem examination all that is observed is that the intestine is distended, and

that its walls are consequently stretched and thin; sometimes the distension is enormous, for the colon has been stated to be as big as a man's thigh. The cause of the dilatation is probably paralysis of the muscular coat of the bowel. The only difficulty of *diagnosis* likely to occur is in the distinction of this condition from those rare cases in which there is gas in the peritoneal cavity.

The *treatment* of tympanites is difficult and uncertain; and its appearance is very unwelcome. Various carminatives, especially the aromatic oils, are usually recommended. They are conveniently given in drops upon sugar, and may perhaps be of some use in gaseous distension of the stomach; but although I have often given them I have never seen them of any use in serious cases of distension of the large intestine; indeed, in some diseases, such as peritonitis and typhoid fever, they hardly seem suitable drugs. Enemas give a better chance of success; the turpentine and asafœtida enemas of the Pharmacopœia are often used; sometimes they certainly afford relief, frequently they fail. In other cases three-quarters of a pint or a pint of soap and water, or half a pint of warm olive oil, may be injected up the rectum, but as with carminatives, so with enemas, we properly shrink from much use of them in typhoid fever or peritonitis. Any of these means may be supplemented by hot fomentations applied to the abdomen. The attempt is often made to draw off the gas from the bowel by passing a long tube up to the sigmoid flexure, but this usually fails; nor is this result surprising when we remember that the aperture in it may be blocked when the tube is in contact with the intestinal wall. It is easier to pass the tube high if the patient lies on his left side.

Puncture of the bowel has been recommended, and this operation is commonly done in veterinary practice. There is a description of it as performed upon sheep in Hardy's *Far from the Madding Crowd.* Bertin (4) mentions a case in which it was performed fifty times on the same patient without the least accident. Still, it is a proceeding which should only be attempted when other means have failed and the tympanites is clearly dangerous to the patient's life. A very fine trocar and canula, which have been previously boiled, should be used; and after the trocar has been withdrawn, and while the gas is escaping from the canula, the abdominal walls should be gently pressed so as to make them follow the intestine as it collapses. The puncture is best made in the middle line, for here the abdominal walls are very thin; and into the transverse colon, as this is usually much distended. The benefit is rarely permanent, for the gas quickly gathers again. Acupuncture should never be employed, as it may lead to the escape of gas into the peritoneal cavity.

(ii.) The second group contains those cases in which the distension of the colon is due to some solid substance within it. In the human subject it is excessively rare for the distending substance to come from outside the body; but concretions consisting of a little fæcal matter incorporated with vegetable fibres, hair, and oat husk have been found, and a good deal of phosphate of lime is usually precipitated from the contents of the

intestine, and this sometimes makes the concretion as hard as a stone. These stony masses are found in the intestines of many vegetable-feeding animals, especially perhaps of horses, cows, and goats. They constitute one variety of bezoards, a name applied to all calcareous concretions found in animals. They are much commoner in the small intestine than in the large.

The substances not introduced from without may be gall-stones or impacted fæces. The former may be as large as a hen's egg, and usually get into the duodenum—or more rarely directly into the colon—by means of a fistulous communication with the gall-bladder. A very good example is recorded by Dr. Wilks (53), in which a young lady, after seventeen days' obstruction of the bowels, passed two gall-stones which seemed to represent a cast of the gall-bladder. The total weight of the stones was 380 grains, and the greatest circumference of the larger was [illegible] inches. Both gall-stones and foreign bodies introduced from without are, however, so rare in the colon that they need not be further discussed. Mr. Eve has collected a series of cases of intestinal obstruction due to gall-stone (17), and Mr. Mayo Robson has recently (39) published a paper on the same subject. In both these papers the operative treatment is discussed.

Distension of the colon by fæcal matter is of great clinical importance as it is of common occurrence. The fæcal mass, which is often of very large size, is usually in the sigmoid flexure or in the descending colon. I have known a mass in this position mistaken for a large tumour of the kidney. The subject of distension of the colon from foreign bodies is fully discussed in the articles "Constipation" and "Intestinal Obstruction."

(iii.) The third group contains those cases in which the dilatation of the colon is due to some organic obstruction in front of the dilated gut. In these the intestine hypertrophies as well as dilates, and the hypertrophy is almost entirely of the circular fibres of the muscular coat; the longitudinal may be somewhat more uniformly spread and more numerous than usual. This muscular hypertrophy may render peristalsis easily visible; but this appearance, though common in the small, is very rare in the large intestine. The intestinal contents retained behind the stricture usually set up some colitis, which may proceed to ulceration, when the well-known "distension ulcers" are produced. This is a bad name, for there is no reason to suppose that the mere distension causes the ulcers. The colitis is shown by swelling and injection of the mucous membrane, and in severe cases the inflammatory process advances so far that the mucous membrane is dark and sloughy. The ulcers may consist of nothing but a slight loss of substance of the epithelial surface; or the whole intestine for several feet behind the obstruction may be covered with irregular ulcers, as was the case with a woman in whom, as a result of malignant disease of the rectum, "the whole of the colon above the stricture was distended and worm-eaten by small ulcers." These, as they increase in size, come to have ragged edges and run one into the other, producing a large area of ulceration. Often the muscular coat is exposed

and sometimes even perforation of the serous coat with consequent fatal peritonitis takes place. The rapidity of this inflammatory ulcerative process must, of course, depend much upon the completeness of the obstruction and the nature of the retained contents. Sometimes it is very rapid, as, for instance, in the case of a woman who was admitted into Guy's Hospital under Dr. Goodhart in 1884 for intestinal obstruction of nine days' duration. At the autopsy volvulus of the sigmoid and acute colitis were found; the distended sigmoid was covered with irregular ulceration on its mucous surface; two of the ulcers were deep and nearly perforating. This case, however, is exceptional, for distension ulcers are commonest in association with chronic obstruction, and are, therefore, most frequently met with behind malignant growths or impacted fæces. The ulcers themselves when exposed appear to have existed for some time, and they may even be pigmented. It is curious to note that they may be a very long way behind the obstruction without any ulcers in the intervening bowel; thus in one case they were found only in the cæcum and ascending colon, although the obstruction was due to carcinoma of the sigmoid. It must not be forgotten that this colitis may, after the obstruction has been overcome, be difficult to treat; and it is the cause of the severe diarrhœa which sometimes follows when after long-standing fæcal impaction the bowels have at last been opened. It will subside, however, under rest in bed, careful diet, warmth to the abdomen, and, if necessary, an occasional starch and opium enema.

The dilatation behind an organic obstruction may be very great, and then it leads to considerable distension of the abdomen. By careful watching of the peristaltic movements, by percussion and by palpation, we can often make out whether it is the large or the small intestine which is dilated; but it must not be forgotten that a dilated small intestine may be as large or larger than the colon, and often lies transversely in the abdomen. On the other hand, a distended transverse colon may bend down towards the pubes like a coil of small intestine, and the sigmoid may be so distended as to reach quite over to the right side of the abdomen. Considerable or even extreme dilatation of the colon is not uncommon in the insane. I need hardly add that slight distension of this part of the bowel often occurs in persons of a constipated habit.

(iv.) The fourth group contains those very rare cases of so-called idiopathic dilatation of the colon. While I was writing this article Dr. Herringham and Mr. Bruce Clarke brought a case before the Clinical Society, and I found that they and I had discovered the same cases scattered through medical literature. The rarity of the condition will be evident from the fact that only about twelve cases can be found in those medical records of the last forty years to which access can be had in London, and, curiously, all these cases are English or American. We have had no case at Guy's since 1880, during which period about 8000 post-mortem examinations have been performed. The reason why it is difficult to be precise as to the number of cases recorded

is that it is doubtful whether some of them should not be regarded a long-standing cases of fæcal impaction. This criticism applies pa ticularly to Dr. Bristowe's first case (6), for the patient, a girl aged years, had always been subject to constipation, and her bowels had n been open for seven weeks before admission. On her death the lar intestine was enormously dilated from the cæcum to a point within 2 inch of the anus, and was completely filled with fæces. Perhaps also Peacock case (37) of a man aged 28, always constipated, who had had a bad atta of fæcal impaction at 17, and in whom hard fæcal accumulation had be broken up more than once, belongs rather to this group. In him also t large intestine was very much distended, being 6 to 8 inches in diamete it contained fifteen quarts of a semifluid greenish fæces, and the muco membrane was much ulcerated. In this case the abdominal distensi "which made him appear like a woman just before her confinemen was associated (with displacement of the heart and liver upwards) wi œdema of the legs, penis and scrotum, and with albuminuria—these l symptoms being apparently due to pressure on the vena cava or re veins.

Putting aside the cases which may have been due to fa impaction, an apparently well-defined group stands out among th which remain, namely, those in which the sigmoid flexure only distended. The following six cases fall under this head:—Mes Herringham and Bruce Clarke (26) give the case of a man over 70 w had always been of an extremely constipated habit. Six years ago had had an attack similar to the present, but it had been overcome strong purgatives. For the last six years he had had little or no trou in this way, but his bowels had now not been open for eight days. T abdomen was distended, enemas and purgatives failed to relieve him, a he died of perforation. The sigmoid flexure was found enormou distended, and resembled a dilated stomach. Its walls were hyp trophied, and its inner surface ulcerated and gangrenous. There w more than one perforation; the rest of the intestine was normal. C stipation lasting eight days can hardly be regarded as sufficient to h produced this condition of the sigmoid. Sir John Banks's (2) patie a man aged 50, was found to have the sigmoid flexure so distended t bent on itself, it filled the abdomen; it was 2 feet in circumference. Chapman's case, a male deaf mute aged 58 (8), the sigmoid bent itself filled the abdomen; its circumference was 14 inches. It actually got up between the right lobe of the liver and the diaphra Money and Paget's case (33) showed the sigmoid to consist of two la sacs even bigger than an ordinary dilated stomach. Dr. Gee's (20) f case was that of a boy aged 4½, in whom the sigmoid was found to c sist of two huge sacs lying vertically side by side, one in the right l of the abdomen, the other in the left. The greatest circumference the sigmoid was 13 inches. His second case, also in a boy, was v similar. The distinguishing characteristic of these cases is that sigmoid is enormously distended, filling the whole of the front of

abdomen. It is in shape like a dilated stomach, and, in Chapman's case, it is stated that the longer curvature measured 20 inches, the smaller 10 or 12. The result of this arrangement is that it is often, as in Dr. Gee's case, described as consisting of two huge sacs lying side by side. The view adopted by some speakers, when Herringham and Bruce Clarke's paper was read, was that a slight accumulation of fæces at one part of the sigmoid leads to kinking of it; that this increases both the obstruction and the accumulation of fæces, and thus one reacts on the other. But it seems to me that this explanation is not certain, and several questions will have to be answered before it is accepted. Why, if there is thus an obstruction at the lower part of the sigmoid, is this part of the bowel so enormously distended and, in these six cases, the rest of it so little distended or not at all? Then, again, these patients were not excessively constipated. For instance, although Herringham and Bruce Clarke's patient had been of a constipated habit, it so happened that before the fatal distension of his sigmoid, "for the last six months he had had little or no trouble, the bowels had been open regularly without more than an occasional mild aperient." When seen he had only been suffering from want of action of the bowel for eight days. Then we must remember that though obstruction about the sigmoid and rectum from fæcal impaction or growth is very common, the condition we are describing is very rare; and from none of the descriptions of the six cases do we gather that the distended sigmoid was full of fæces: in fact, it is quite clear, from some of the descriptions, that this was not the case. Then in some instances, even shortly before death, the obstruction was not complete, for in Dr. Gee's first case there was diarrhœa up to death. It is, I think, more probable that this local dilatation of the sigmoid is comparable rather with the cases of dilatation of the stomach without obstruction of the pylorus. And I notice that Rolleston and Haward, in a paper published after I wrote this account, adopt the same view. The one is often associated with dyspepsia, the other with faulty action of the bowels; but in both the precise relationship of the perverted action to the dilatation is obscure, and in neither is it easy to be sure of the cause of the condition. Why the sigmoid should be more affected than any other part of the bowel is not clear to us; but it must be remembered that, according to some authors, as to Trastour (45), it is the habitual storehouse for fæces between the acts of defæcation, and that slight dilatation of it is common.

The following cases, in which the dilatation was near the sigmoid rather than of it, probably belong to the same clinical group:—

The first, recorded by Messrs. Walker and Griffiths (47), was that of a boy aged 11, whose abdomen was enlarged and tympanitic soon after birth; his bowels were regular at first, but afterwards he suffered from constipation, although they could always be opened by enemas, and it does not appear that there was ever any obstruction. His appetite was bad, his breathing was short, wind would occasionally "roar away," and then the distension lessened. So distended

was he, that although his height was only 4 ft. the abdomen measure 23 inches from the ensiform cartilage to the pubes, and its greatest girl was 3 ft. 11 inches. On opening the abdomen after death, it appear filled with an enormous coil of intestine, looking like a flexed thigh a leg, formed by the transverse and descending colon, the latter 23 inches i circumference. The sigmoid and ascending colon were somewhat enlarge The floor of the thoracic cavity was so pushed up that it resembled shallow pie-dish; from the top of the diaphragm to the suprasternal not was only 2½ inches. The peritoneal coat of the affected bowel was thi the muscular coat was hypertrophied, and there was no submuco tissue; the mucous membrane was thick, often denuded of its epitheliu and in a state of chronic inflammation.

The second case, also in a boy, was recorded by Dr. Rolleston and Warrington Haward (41). He was twelve years old at death, and had suffered from attacks of constipation and vomiting together w a distended abdomen since the age of two months. The descending co and the cæcum were enormously dilated.

Another case is recorded by Messrs. Little and Callaway (3 The patient was an imbecile male aged 34, who had alw had an inordinate appetite; and after eating an unpeeled ora and a pound of Banbury cakes was taken with abdominal pain sickness. Aperients acted, but the next day extreme disten was noticed, the circumference at the umbilicus being 45 inches. heart was very much displaced, and he sank on the seventh day. Th was enormous dilatation of the transverse and descending colon, and the sigmoid, the muscular and mucous coats of which were very thick

The fourth case is recorded by Dr. Goodhart (23), and prob Dr. Herringham is right in classing it with these cases, althoug had previously associated it with ulcerative colitis. The patien girl aged 17, had noticed abdominal distension for eight mon during which time constipation alternated with diarrhœa. She died f perforation. The large intestine was dilated from the cæcum to anus, but the dilatation was not local, as in the other cases, nor wa so extensive. There was a considerable amount of ulceration. W (49) gives a case of an elderly man in whom the transverse colon distended in the form of a loop, but it is just possible that s adhesions of the small intestine were the cause of this.

In Bristowe's case of a male, aged 24, there was so m diarrhœa and so much ulceration, that it is very doubtful whethe should be included in this category; I shall therefore leave it out of sideration.

Osler's case (35) forms a connecting link between the group of dil tion of or near the sigmoid and those of dilatation of other parts of the co The patient, a coloured boy aged 10, was thin, but enormously disten in the abdomen. He was not particularly constipated, and often diarrhœa and vomiting. Intestinal peristalsis could be seen. For a t washing out the bowel with water, introduced with a long tube when

hips were elevated, led to some relief; but this was slight, and the pain and vomiting continued. The abdomen was opened. It was clear there was no stricture. The sigmoid was 18 inches in circumference, the cæcum was half this size, and the bowel progressively increased in size from the cæcum to the sigmoid, which was folded on itself, but not so as to cause any obstruction. An artificial anus was made at the most prominent part of the sigmoid. The mucous membrane seemed normal, but the muscle was hypertrophied. After the operation his appetite and general condition improved, he had no tympanites, he gained in weight, and passed the fæces through the artificial anus.

These cases are too few for generalisation, but it is noteworthy that out of the twelve I have mentioned eleven were in males, and the one in a female is a doubtful example. This tells against the view that this condition is due to chronic constipation; for women are more liable to this than men. The cases appear to fall chiefly into two groups, for Herringham and Bruce Clarke's, Banks', Chapman's, Money and Paget's, and Wells' cases were men over 50; while Gee's two cases, Walker and Griffiths', Rolleston's and Haward's, and Osler's were children, who had probably suffered from abdominal distension since birth. Goodhart's and Little and Callaway's cases do not fall into either group. It seems probable, therefore, that we shall have to distinguish two varieties of so-called idiopathic dilatation of the colon; namely, that occurring in elderly subjects, and that, apparently due to some congenital inertness of the bowel, met with in young children. The chief symptoms of this condition, as exemplified by these eleven cases, are as follows:—

Symptoms.—The distension of the intestine is very great, and percussion shows that it is chiefly due to gas. Shortness of breath is a cause of complaint, and the patient may be livid, because the diaphragm is pushed up and breathing is hampered. The upward displacement of the heart gives rise to palpitation. The splenic dulness is obliterated, the hepatic dulness is diminished, and the liver cannot be felt. The abdomen is always distended, sometimes enormously so, and then it may be tympanitic all over; but neither distinct coils nor peristaltic movements are usually visible. In Money and Paget's case the intra-abdominal pressure was so great, that the legs, scrotum, and penis were swollen; the renal veins, too, were probably compressed, as the patient had albuminuria. After the gas had been let off by puncture of the intestine the albuminuria passed away, the specific gravity of the urine fell, and the patient passed eight pints of urine in the night. Dr. Gee's first case had hæmaturia shortly before death. Most of the patients for some time, often for years, have had some difficulty with the bowels, constipation supervening from time to time; but this is not usually very serious, and has yielded to purgatives or enemas. In Money and Paget's case the bowels had always been regular; and for some years before the fatal attack they had been so in Herringham and Bruce Clarke's case. Occasionally diarrhœa sets in shortly before death. The condition found after death has already been indicated in the description of the individual

cases. It will be noticed that both layers of the muscular coat of the are hypertrophied, and its mucous membrane often ulcerated, prob from the colitis set up by the irritation of the decomposing fæcal ma retained in the distended bowel. Except for the fact that the kid may be hard, like heart kidneys—probably owing to compression of renal veins—the whole body, save the colon, appears healthy; an no case has any obstruction been found in the intestine. The condi appears, therefore, to be a purely local one. The distended b contains semi-solid fæces and an enormous quantity of gas. It been found on measurement to be increased in length as well a diameter. All the patients except one died. In two the cause of d was perforation of one of the ulcers, but in none of the others was cause of death clear. Often it took place a few days after the pa was first seen, and with some it was unexpectedly sudden.

Enemas and purgatives commonly had no effect; and even if bowels were opened the patient was not much improved thereby. passage of a long rectal tube often failed to bring away any gas, although sometimes this proceeding or puncture of the intestine reli the pressure for a time, the gas soon reaccumulated. As these result so poor I would suggest that the best chance is afforded by opening bowel above the dilated part; and as this part is usually the sigmoid t would be very little difficulty in the operation. For some time the f should all be passed through the artificial anus, and the distended sig should be thoroughly flushed from the artificial opening to the every day with clean tepid water. It seems quite hopeless to ex the bowel to recover itself so long as fæces are continually passed it. So much improvement followed in Osler's case, in which the sig was opened, that we ought to be encouraged to adopt the treatment suggested.

IX. Sacculation of the colon is not a common condition, but one who has made a considerable number of post-mortem examina is sure to have met with examples of it. The diverticula are usually size of a pea, but I have seen them big enough to receive the little fi and half an inch in depth. They are most frequent in the descen colon, sigmoid flexure, or upper part of the rectum. Usually the very numerous, are placed closed together, and contain a little f matter. They are described as hernial protrusions of the mucous m brane through the muscular coat, and no doubt this suggestion is corr but often a thin layer of muscle is spread over the whole surface sacculus except just at its bottom, which consists of mucous memb and peritoneum only. Sometimes, as in a specimen I showed at Pathological Society, these diverticula lead into appendices epiploicæ (and Wilks and Moxon mention a case in which they protruded betwee layers of the mesentery. Perhaps they should be regarded as du obstruction in the form of chronic constipation; and in favour of this is the fact that they are found for the most part in elderly people, an far as I know, never in children. As a rule they give rise to no sy

toms; but we have in the Museum at Guy's Hospital a specimen in which perforation of the fundus of one of these sacculi led to the formation of an abscess between the left kidney, spleen and ascending colon.

Solitary diverticula are very rare; but in 1888 an autopsy was made at Guy's Hospital on a patient who died of malignant disease of the œsophagus; and in the colon ten inches from the ileo-cæcal valve was a diverticulum half an inch long which admitted a No. 10 catheter.

This is perhaps the best place to mention that the appendices epiploicæ may become pedunculated, and enlarged at their lower part; so that they really form subserous lipomas. This is more common in connection with the colon than with other parts of the intestine. Small pedunculated lipomas may become detached, and may be found free in the peritoneal cavity. They are sometimes calcified.

X. Laceration of the colon. — Sometimes, but happily very rarely, this is due to an enema tube or a bougie. We have two such specimens in the Guy's Hospital Museum. In the first case an enema was given by means of a long tube to a man aged 75; shortly afterwards he was admitted to the Hospital collapsed, and he died the next day. A laceration was found at the lower end of the sigmoid flexure; the perforation in the mucous membrane was only one-eighth of an inch in diameter. The other specimen, presented by Dr. Hodgkin and Mr. Callaway, represents a condition that could hardly occur nowadays. It consists of the sigmoid flexure with a rent more than an inch long, due to a bougie which had been passed, under the impression that the patient had stricture of the rectum; he was really suffering from fæcal impaction.

It is not very infrequent for a nurse or a doctor to be excessively alarmed by the apprehension that the perforation of the rectum or sigmoid has been produced by an enema—as in elderly people the evacuation of the bowel by an enema is often accompanied by much faintness and even by syncope. The pulse may become quite small, and the patient appear collapsed. I remember being summoned very urgently to see an old lady in this condition, who had been allowed to get up to go to stool after the enema. This should never be permitted in elderly or feeble people who are unaccustomed to enemas. In fact, constipation in such persons is to be specially avoided. If, however, syncope does occur, the treatment is the same as for that due to any other cause.

The kinds of accident that most frequently damage the intestine are, being run over, kicked in the abdomen, or stabbed there. The small intestine is much more often affected than the large; but a few years ago a man who had been run over was admitted into the Hospital, and the only lesion found was a large tear in the ascending colon. The laceration may not go quite through, as in the case of a man who was run over by a brewer's dray. The small intestine was torn completely across, but in the descending colon the muscular and serous coats were extensively lacerated, although the mucous membrane was entire. We have a specimen

also in which the muscular coat only is ruptured, and another in whic the serous alone is affected.

XI. Perforation of the colon from without. — Abscesses in variety of positions may occasionally burst into the colon. Thus bullet entered the abdomen from behind; a large retro-peritone abscess formed and opened into the descending colon. Instances iliac and psoas abscesses rupturing into some part or another of t colon might be quoted; and a suspicion of what has happened may afforded by the disappearance of the tumour and the passage of p by the bowel. I have come across two cases, both in women, in which t localised collections of pus, which occur in suppurative peritonitis wh adhesions are numerous, had burst into the colon. In both cases th had been many perforations, and in both the opening inwards of t abscesses had caused a number of clean-cut circular ulcers, on the fl of which was a perforation. In the second case there were other ulc as well, so it is probable that the presence of pus in the colon set colitis followed by ulceration. Recently I have had a young woman un my care in whom a localised peritoneal abscess was found pointing in t inner surface of the stomach; in a day or two it would have formed ulcer there with a perforation on its floor.

As the liver lies over the hepatic flexure of the colon hep abscesses may burst into the bowel. Murchison (34) gives a case which a large abscess opened freely into the colon, and there was tensive ulceration of the adjacent portion of the ascending colon. T is important, as, like one case just mentioned, it goes to show t pus discharging into the bowel can set up ulceration of it; but still i possible that this case was one of ulcerative colitis, associated wit large hepatic abscess. He also records a case in which a free discha of pus by the bowel showed that a hepatic abscess had burst into but the abscess also formed a bulging of the abdominal wall; this incised, the abscess thoroughly drained, and the patient made an excell recovery. In rare cases abscesses formed in connection with the pendix, and those arising in or around the kidney, may open into colon; and Dr. Bright (5) has recorded the case of an abscess of the spl discharging into the colon. Dr. Habershon mentions two remarka cases of cystic ovarian tumours: in one the tumour opened into ileum, and thus became filled with fæces; in the other it suppurated, ruptured both externally and into the cæcum, so that there was a charge of fæces externally. Fagge mentions a case of suppuration ab a growth in which the same thing happened; and Habershon gives instance in which an abscess in the abdominal parietes burst b externally and into the colon, and so formed a fistulous communicat into the large intestine.

Hydatids of the liver may be discharged into the colon, and tl cysts may be found in the evacuations. The gall-bladder may adh to the hepatic flexure, and large gall-stones may ulcerate their way i the bowel. Occasionally in cases of ulcer of the stomach, either sim

or carcinomatous, this organ may become adherent to the transverse colon into which the ulcer perforates, and thus a gastro-colic fistula is formed. In 1887 this condition was found in a woman who died from chronic bronchitis. As far as was known it gave rise to no symptoms, but cases have been recorded in which such a fistula has caused the vomiting to be fæcal.

XII. Malformations of the colon.—Malformations of the colon are rare, but by far the most common are those which depend upon malpositions of the cæcum. It will be remembered that in intra-uterine life this is first outside the abdomen, then inside just under the umbilicus, which is then near the pubes. In its next position it is on the left of the abdominal cavity; its last position on this side is near the cardiac end of the stomach; then it passes across the abdomen and comes to lie under the liver, and finally it descends to the right iliac fossa. The cæcum in an adult may be in any of its successive intra-abdominal positions, from just inside the umbilicus to the normal one in the right iliac fossa. It is quite possible that in some cases the persistence of the cæcum in a part of the abdomen, which in the natural course of events it should have left before birth, is due to fixation from intra-uterine peritonitis—a disease by no means uncommon, especially in children afflicted with congenital syphilis. When the cæcum is fixed in its abnormal position the large intestine continues to grow, and consequently various abnormal loops are formed. As these conditions have been known to give rise to difficulties both to the physician and to the operating surgeon, they should never be forgotten in considering a case which is manifestly obscure. Various references to actual cases will be found given by Mr. Lockwood (32), who points out that the failure of the cæcum to descend is often associated with an undescended testis. Other malformations are excessively rare. Lockwood quotes two cases in which, for part of its length, the large intestine consisted of two tubes; and we have in Guy's Hospital Museum a specimen taken from a boy aged seven days which shows a membranous septum completely occluding the bowel at a point nine inches above the anus. The colon was much dilated above; below, the gut was contracted to the size of a cedar pencil.

XIII. Metallic pigmentation of the colon.—Sometimes lead may be deposited in the tissues of the colon. The three following instances have occurred at Guy's during recent years:—A man who had lead colic died in 1888, under the care of Dr. Pye-Smith, from chronic Bright's disease. The whole of the cæcum and large intestine was very black, and the line of demarcation at the cæcum was quite sharp. Dr. Stevenson found the colon to contain 0·0086 per cent of lead. Dr. Pitt has recorded the case (38). Another man was admitted in 1890 for saturnine epilepsy; he died, and the colon was found very black from lead. Microscopical examination showed minute black granules arranged just outside the vessel in the vascular loops. A woman who was under my care in 1892 for lead colic died from saturnine eclampsia. At the ileo-cæcal valve there was a sharp line of demarcation beyond which the colon was

almost black as far as the sigmoid. The pigmentation was most mar at the mouths of the follicles, and was in places patchy.

There is nothing to show that lead is not deposited in the colon the same process as leads to the formation of a blue line on the g and around the anus.

Two cases of black colon from mercury occur in the *Patholo Society's Transactions.* One in vol. xviii. is recorded by Dr. C Williams. The patient, a lady aged 74, took eight grains calomel a week all her life. At the autopsy the internal surface of large intestine was remarkably black in patches. This discolor began abruptly at the ileo cæcal valve. Analysis showed the presen mercury in the mucous and submucous coats. Another is recorded in xliii. by Dr. Rolleston. The patient, a man aged 64, had un gone antisyphilitic treatment for some time, how long is not quite cer The cæcum and colon were uniformly pigmented. There was no pigm tion of the small intestine. Analysis showed the mucous and submu tissues to contain mercury.

The colon may be found blackened from bismuth in patients who taken much of it during life, and probably, like mercury and lead, deposited in the tissues of the large intestine; but analyses on this su are wanting. Bismuth also, like iron, may darken the fæces.

XIV. Malignant disease of the colon.—A primary growth in colon is almost always a cylindrical-celled epithelioma, usually cir scribed to a small spot, and by its contraction setting up an an stricture there. This causes intestinal obstruction and leads to dilatation of the colon behind (p. 966) the stricture, with, probably tension ulcers. At death, sufferers from this form of carcinoma us present secondary nodules in the liver which exactly resemble the ori growth. As a patient afflicted with malignant disease of his colon n always suffers from intestinal obstruction, malignant disease of the is for the most part described under this head (see p. 827). Occasion however, the growth is soft and medullary; then it breaks down i centre and intestinal obstruction does not arise. A post-mortem exa tion in these cases reveals that several inches of colon are affected growth, usually forming an obvious tumour visible directly the abd is opened. On cutting into this mass it is found that the normal ch of the intestine is replaced by an elongated cavity with black, ra sloughy walls, but passing into healthy colon both above and belo have known the whole of the transverse colon to be converted such a mass. The contents of the cavity consist of a foul mi of fæces, blood, and necrotic fragments of growth. If the sigmo cæcum is affected this mass may grow into the subjacent bone; if in other parts of the colon it may extend into the stomach, liv kidneys. This state of things during life may give rise to no symp as in the case of a woman who recently died under my care from c of her liver. During life we could nowhere find the primary seat o growth, but at the autopsy it turned out that the sigmoid flexure

sloughing carcinomatous mass. On the other hand, it may be easy during life to detect a tumour, especially if it is in the transverse colon or the cæcum. The patient may pass large quantities of foul, dark gray or black fluid motions, in which perhaps fragments of growth and small blood-clots are seen. Sometimes between the passage of such motions the bowels may be constipated. The patient suffers pain, he wastes, and I have known death to occur in such a case before any symptoms of secondary deposit in the liver showed themselves. When the tumour is in the cæcum or sigmoid it may cause œdema of one leg and thrombosis of the external iliac vein, and when it is in the transverse colon we must carefully distinguish it from an enlarged liver, carcinoma of the greater curvature of the stomach, or an omentum puckered up by some form of chronic peritonitis. The implication of other organs by direct spread of the growth rarely gives rise to any symptoms by which it can be recognised. In very rare cases of carcinoma of the large intestine a fistulous tract is formed which opens either into the stomach, the duodenum, or some adjacent coil of small intestine. In the Museum of Guy's Hospital there is a specimen which shows carcinoma of the sigmoid flexure which formed a fæcal fistula that opened externally. In rare instances the carcinomatous growth ulcerates directly into the peritoneal cavity.

It is obvious that in most instances the only treatment for these cases, in which there are no signs of obstruction, is to deal with symptoms as they arise. It is very rarely that the case is seen early enough for the surgeon to be able to excise the growth.

Occasionally after death it is found that these cylindrical-celled cancers are villous; or more commonly they have undergone colloid degeneration. Spheroidal carcinoma is rare in the large intestine; we have seven specimens of it in the Museum at Guy's Hospital; but it is rarer still in the small intestine, for we have only one in which it was situated there. Of the seven cases the growth was in the cæcum in two, at the commencement of the large intestine in one, in the transverse colon in two, in the splenic flexure in one, and near the sigmoid in one. In three cases it had undergone colloid degeneration. As in the cases in which the growth was a cylindrical-celled carcinoma, often the cases were first brought under the observer's notice on account of intestinal obstruction.

Sarcoma (see also p. 831) of the colon is very rare; it may be either round-celled or spindle-celled. Lymphadenoma of the intestine is incidentally mentioned in the article on "Tumours of the Stomach," p. 554; it is excessively rare in the large intestine, but we have a specimen in which it occurred as a part of generalised lymphadenomatous growth in Hodgkin's disease. A mass the size of a man's fist was growing in the cæcum, and numerous masses projected from under the mucous membrane in the colon.

Secondary deposits are occasionally seen in the colon, usually on its peritoneal surface; they rarely give rise to symptoms. Malignant disease may spread from the stomach to the colon, as described in the article on "Tumours of the Stomach," p. 554. The inroads of cancer from other organs are of little interest in this place.

Polypi are not uncommon in the colon, but they are of no importance. These and other innocent tumours are described on

Specific diseases (as dysentery, typhoid, etc.), in which the c affected, will be found under their appropriate headings. Lar disease of the colon does not differ from that of the small intestin

W. HALE WH

REFERENCES

1. ACLAND. *Path Trans.* vol. xxxvi. p. 56.—2. BANKS, Sir JOHN. *Dublin Q Journal*, 1846.—3. BANNATYNE. *Edin. Med. Jour.* Aug. 1894.—4. BERTIN. *naire encyclopædique des sciences médicales*, t. xix.—5. BRIGHT. *Guy's Hospital* 1838.—6. BRISTOWE. *Brit. Med. Jour.* 1885, vol. i. p. 1085.—7. BRUNNER. *Rev.* Sept. 1892.—8. CHAPMAN. *Brit. Med. Jour.* 1878, vol. i. p. 566.—9. COLMA *Guy's Hospital Gazette*, 1893.—10. COWAN. *Lancet*, March 16, 1895.—11. SON. *Path. Trans.* vol. xxxii.—12. *Idem. Med.-Chir. Trans.* vol. lxxvii.— COSTA. *Amer. Jour. Med. Sci.* vol. lxii. p. 338.—14. EDWARDS. Keating's *Cy of Diseases of Children*, vol. iii.—15. EICHHORST. Eulenberg's *Real-encyk* Bd. v. S. 344.—16. EURICH. *Lancet*, May 18, 1895.—17. EVE. *Clin. Soc.* 1895.—18. FAGGE. *Path. Trans.* vol. xxi.—19. FINLAY and ADAMI. *Montr Jour.* April 1894.—20. GEE. *St. Barth. Hosp. Rep.* vol. xx. p. 19.—21. G BIRD and W. HALE WHITE. *Clin. Soc. Trans.* vol. xxix. 1895.—22. Goo *Path. Trans.* vol. xxxvi. p. 203.—23. *Idem. Clin. Soc. Trans.* 1880.—24 *Hosp. Rep.* vol. i. p. 382.—25. *Ibid.* vol. xlv. p. 145.—26. HERRINGHAM and CLARKE. *Brit. Med. Jour.* 1894, vol. ii.—27. HOFFMANN. *Wiener med* 1872, No. 24.—28. HOLT. Keating's *Encyclopædia of Diseases of Childr* HOWSE. *Path. Trans.* 1878.—30. LIGHT. *Practitioner*, 1893.—31. LITTLE a LAWAY. *Path. Trans.* vol. iii. p. 106.—32. LOCKWOOD. *Brit. Med. Jour.* 18 MONEY and PAGET. *Clin. Soc. Trans.* vol. xxi. p. 103.—34. MURCHISON. *of the Liver.*—35. OSLER. *Johns Hopkins Hosp. Rep.* Bulletin iv. 30.—3 *Trans.* vol. xxxiv.—37. PEACOCK. *Path. Trans.* vol. xxxiii.—38. PITT. *Path* vol. xlii.—39. ROBSON, MAYO. *Clin. Soc. Trans.* vol. xxvi. p. 213.—40. *Idem. Chir. Trans.* 1895.—41. ROLLESTON and HAWARD. *Clin. Soc. Trans.* vo 1895.—42. SIMPSON. *Med. Press and Circular*, July 29, 1896.—43. SMITH, S *Jour.* Sept. 5, 1894.—44. TARGETT. *Path. Trans.* vol. xliii.—45. TRASTOUR. *mensuelle de méd. et de chir.* 1880.—46. VIRCHOW. *Berl. klin. Woch.* 18 50.—47. WALKER and GRIFFITHS. *Brit. Med. Jour.* vol. ii. 1893.—48. WASI *Med.-Chir. Trans.* 1894.—49. WELLS. *Transactions of a Society for the Imp of Medical and Chirurgical Knowledge*, 1812.—50. WHITE, W. HALE. *Guy's Reports*, vol. xlv. 1888.—51. *Idem. Path. Trans.* vol. xxxvi. p. 215.—52 *Lancet*, 1894.—53. WILKS. *Path. Trans.* vol. xxxi. p. 218.

W. H.

THE DIFFERENTIAL DIAGNOSIS OF DISEASES OF THE ANUS AND RECTUM

THESE diseases, strictly speaking, belong to the sphere of the surgeon. Nevertheless, the physician is often consulted in cases of alleged diarrhœa, in which an exploration of the rectum may be of the first importance; or the presence of piles may hinder the cure of cases of anæmia or other ailments which properly fall to the care of the physician.

The maladies which affect the anus and rectum are of great variety and are widely prevalent; they cause the sufferer much pain and discomfort, and are the source both of local annoyance and of mental distress. Nevertheless, in spite of the importance of the subject, the diagnosis of anal and rectal diseases has not received the great attention which is due to it. Various reasons appear to deter some medical men from investigating these ailments with sufficient care, especially when the patient is a woman. Thus no examination is made at all, or but a very perfunctory one. The consequences of this neglect would be serious enough if there were but one rectal malady; they are intensified by the fact that the diseases affecting this small portion of the human frame are many.

A careful local examination is absolutely necessary; first of all to arrive at the cause of the suffering, and then to determine on the right line of treatment to be employed for the cure of the particular malady. It is one of the greatest mistakes in medical practice to imagine that *Unguentum gallæ cum opio* and *Mag. sulph.* are the panacea for all rectal troubles. Each disease requires its proper mode of treatment, otherwise there will be no cure; indeed the patient may be made still worse.

But if the diagnosis is correctly made and appropriate treatment used, whether medicinal or operative, an ordinary case of rectal disease can be cured readily and with little fear of a recurrence. Unfortunately, such success will never be attained so long as all rectal troubles are ascribed to piles; indeed, in some instances medical treatment for piles has been pursued when the patient was actually suffering from malignant disease. For such shameful carelessness no censure can be too strong. Again, even if the ailment be piles which are continually bleeding, patients are sometimes told that the hæmorrhage will do them good; though they may be gradually growing weaker from more or less loss of blood. This habit of regarding piles as the one malady of the rectum is found also in our patients when they come for consultation. Patients tell us that they are suffering from this complaint; their own diagnosis is too often accepted, and a thorough examination is postponed, with consequences more or less serious if the case actually be one of pruritus, fissure, polypus, fistula, ulceration, or even of cancer. We can only arrive at a correct

diagnosis by asking the patient first of all the following questions; further interrogation may follow a careful local examination:—

(*a*) "Have you pain? If so, where and when?"

Affirmative answers to this question may refer to fissure, ulceration, abscess, or malignant disease respectively.

(*b*) "Is there any discharge? Of what nature is it? Is it pus, mucus, or blood?"

The discharge of pus will confine the diagnosis to abscess, fistula, and ulceration.

Mucus will suggest piles, ulceration, or malignant disease.

Blood may be discharged in any rectal ailment, and is therefore of no aid in diagnosis.

(*c*) "Is there any protrusion at stool which must be returned by pressure?"

A protrusion may arise from piles, polypus, or polypoid growths.

(*d*) "Is there diarrhœa or constipation? If diarrhœa, is it in the early morning on first rising? Is any blood or slime passed with the stools?"

Diarrhœa will indicate ulceration or cancer; morning diarrhœa will point to cancer in particular.

From the replies to the above questions we may easily obtain some idea of the nature of the disease.

The next step is a thorough examination. There are various positions for this; and if the case be a difficult one to diagnose, the patient may be required to kneel or even to stand up. But the best, and at the same time the most delicate posture is for the patient to lie on the right side, with the knees fully drawn up to the abdomen and the buttocks well to the edge of the couch, and for the examiner to kneel on the floor facing the buttocks. The patient should be asked to hold up the upper buttock with the left hand so as to give a good view of the anus and the surrounding parts.

The medical adviser should then look carefully round the parts. His first attention should be turned to the condition of the skin of the anus, to see whether it is cracked, inflamed, or eczematous; or whether there are any scratch-marks. He should note the shape of the anus, and observe whether it is pouting, appearing to be tightly contracted, and so forth. Moreover, he should not fail to look for any orifices of fistula, and should not disregard the presence of any excessive amount of loose skin around the verge of the anus.

This ocular examination completed, with the forefinger of the right hand he should diligently feel round the anus for at least two inches from its orifice for the purpose of discovering any tender spot. Drawing the finger over the parts, he should press deeply in order to detect any induration pointing to the site of an abscess, or any cord-like ridge running up towards the anus, indicating the track of a fistula. The pressure may cause pus to ooze from the sinus of a fistula, or from the anus itself. Any small folds of skin must be carefully separated; for between or be-

hind them the orifice of a fistula may be situated, and this may be easily overlooked. When any of these abnormal conditions round the anus has been observed or felt, the next step is to separate the verge of the anus upwards and downwards with the fingers of both hands. At the same time the patient should be directed to strain down, as this action will give a view of the interior of the anus for about half an inch. In this manner a fissure may be noted and can be readily examined; the situation of a fissure in the large majority of cases being in the middle line behind: or again piles or a polypoid growth may protrude.

Then follows the most important point of a thorough examination, the insertion of the finger into the bowel. The finger, well lubricated with some unguent, is gently inserted, the patient straining down as before. This act releases the sphincters and renders the insertion more easy. At first merely the tip of the finger is placed within the entrance, and the breadth and tightness of the external sphincter are carefully ascertained. Then the finger is inserted a little farther and a diligent scrutiny is made of the interval between the two sphincters; it is in this space that piles are to be felt as enlarged rectal folds; and here, too, in at least 90 per cent of cases, the internal openings of fistulas are found as small dimples or as areas of induration. These internal orifices of fistulas are as a rule not more than an inch from the anal margin; it is altogether a mistake to suppose that their situation is high up the rectum. Again, between the sphincters we may find polypoid growths, the frequent cause of fissure. Unless these are discovered and removed it is useless to treat a fissure, as they will prevent it from healing.

When this area has been explored the finger is passed a little higher up the rectum, and the internal sphincter is searched for any induration which might point to the existence of an internal fistula, or of an abscess. Last of all the finger is pushed to its full extent into the bowel and the higher parts are carefully examined. Several points are to be noted, namely, the prostate gland towards the front, or the uterus which is easily to be felt. Or again, we may discover a malignant mass projecting into the upper part of the rectum, and this will give the finger the same sensation as is caused by the os uteri when felt from the vagina. If the symptoms appear to indicate a polypus the finger must be gently swept round the bowel; by this plan the stalk may be felt and the polypus itself may be brought within reach. When the finger is withdrawn, note should be taken of the character of any discharge left upon it, such as fæcal matter, blood, pus, and so forth.

When this thorough examination has been made with the finger, starting from the outside and passing upwards as high as possible in the order described above, and when all the different points with regard to normality or abnormality of state have been carefully observed, something further may still be needed to confirm or to complete the diagnosis. If about or near the verge of the anus there is a suspicious orifice, suggestive of a fistula, a probe may be inserted with the greatest gentleness into this orifice, made to find its way quietly along the hardened ridge

towards the anus, and thus passed into the bowel; unless the sinus be too tortuous. Again, with the patient straining down, and the physician everting the anal margin with the fingers, a probe may be used gently to explore any ulcer or fissure, in order to discover whether there be any burrowing backwards and outwards, or even running up the bowel from the fissure. This burrowing often exists, and if it is not detected the patient will not be cured either by the use of ointment, or by the division of what is presumed to be a fissure, but which is in reality a fissure with a small sinus running from it. The probe may be employed in a similar manner if the finger light upon an orifice situated between the two sphincters. Should there be an induration around the anus, which would prove this orifice to be the opening of a fistula, there would be no necessity to use the probe; but if there be no such induration, the probe must be introduced along the finger of the left hand as far as the orifice, and then passed into it to discover any sinus which starting from it may run up the rectum.

It is possible that all this examination with the finger, and perhaps with the probe, may have revealed nothing; but the symptoms may appear to indicate some disease higher up the bowel, namely, the presence of a growth, or of one of the forms of ulceration combined with stricture. With cancer the diagnosis will be certain, for in that case there is always a hard ulcerated growth. There will be more difficulty in distinguishing between the various kinds of ulceration combined with stricture; such as syphilitic, tuberculous, traumatic, dysenteric.

The history alone will enable a correct diagnosis to be made, for these diseases present precisely the same appearance, give the same "feel" to the finger, and have identical symptoms; namely, morning diarrhœa, the passage of blood, jelly-like discharge, pain and tenesmus. Moreover, they may each of them involve portions of the rectum or even the whole of it. If the condition be syphilitic there will be a marked syphilitic history. If it be tuberculous, there will be the history of this complaint, and further, a bacteriological investigation of the pus may show tubercle bacilli. If the case be traumatic the patient has probably suffered from a very prolonged and severe confinement. In the dysenteric cases it will be found that the patient has resided in foreign parts and has there contracted dysentery.

In cases of suspected disease higher up in the bowel, the diagnosis may be assisted by the passage of bougies, or of a bougie à boule. By the use of these one may explore the upper parts of the rectum and the lower end of the sigmoid flexure. Note should be taken of the respective degrees of ease with which the bougies are passed, and of the nature and position of any discharge which may come away upon it.

Attention should then be turned to the abdomen, which should be examined with care, especially the left iliac fossa. The hand should be pressed deeply into it, and note should be taken of any tenderness, or of any tumour which may be made out.

Yet, even when all these methods of scrutiny may have been pursued,

a sure diagnosis may still be wanting. When this is the case the patient must be anæsthetised and the examination must be carried out again in the various ways described above. For, when the patient is under the influence of an anæsthetic, the sphincters can be thoroughly dilated and the upper parts of the rectum can be more completely searched. Nor need we now confine ourselves to digital examination; the gut itself can be seized and pulled down so as to be brought into full view.

It will be observed that no mention has been made of the employment of the speculum. Under an anæsthetic the speculum may be of service; but in all other cases this instrument, for purposes of rectal examination, is useless if not harmful. To be of real service the speculum must be large, but if large it will cause the patient great pain. In all cases but those of very great obscurity, a systematic examination with the finger and probe will discover all that is necessary for correct diagnosis.

HERBERT WM. ALLINGHAM.

LIST OF AUTHORITIES

ABELES, 204
Abelmann, 301
Abelous, 293
Abercrombie, 517
Acland, T. D., 963
Adami, 675
Adams, R., 80
Addison, T., 217
Albarran, 606
Albers, 879
Alderson, 450
Allbutt, Clifford, 78, 362, 443, 577, 659
Allchin, 713
Alt, 415
Anderson, 676
Andral, 442, 492
Andrew, J., 26
Annesley, 776
Apt, 208
Aretæus, 195
Armanni, 209
Arthus, 203
Ashby, 23
Audry, 337
Austen, J. A., 11 (*note*)

BABES, 4, 726
Baginsky, 130, 643
Baillie, M., 14 (*note*), 517
Baker, Morrant, 68
Balfour, 296
Ballance, 328
Ballantyne, 75
Ballard, 747
Ballonius, 1 (*note*)
Bamberger, 501, 517
Banks, John, 968
Bannatyne, 957
Banti, 59, 107
Bard, 720
Barker, Fordyce, 454
Barling, 547
Barlow, 872
Barlow, Lazarus, 324, 956
Barlow, T., 29, 47, 59, 104 (*note*), 112 *et seq.*
Barnes, 449
Barth, 711
Basch, von, 695
Bauer, 635
Baumel, 208
Bavay, 777
Baxter, B., 123
Beale, 208
Beard, 450
Beaumont, 390, 418
Beck, 204
Bednar, 432
Begbie, Warburton, 76, 219, 405
Begg, 792
Bennett, W. H., 515
Berg, 337
Berger, 849
Bergeron, 334
Bergmann, von, 364, 427
Bernard, Claude, 200, 282
Berry, J., 376
Bertillon, 197
Bertin, 964
Bertrand, 776 *et seq.*, 955
Besnier, 58
Bird, Golding, 711, 947
Bizzozero, 432
Blackadder, 885
Blake, John, 867
Blaxall, 75
Blumer, 71
Blyth, Wynter, 786
Boas, 290, 492, 517
Boetcher, 526
Bohn, 336
Booker, 748
Bordoni-Uffreduzzi, 69, 71
Böttger, 214
Bouchard, 491
Bouchardat, 224
Bouillaud, 19, 45
Bouveret, 427, 501
Bradford, Rose, 179 (*note*)
Brandes, 66
Braun, 214, 876
Bremer, 211
Bret, 442

Endmann 492
Erb, 112
Escherich, 605, 747
Esmarch, 20
Eurich, 964
Eve, 966
Ewald, 288, 422, 501, 517, 559, 709
Ewart, W., 515

FAGGE, HILTON, 91, 174, 197, 326, 488 *et seq.*, 635, 730, 810 *et seq.*, 958
Fayrer, Joseph, 415, 743, 776
Fenwick, Samuel, 422, 680
Fenwick, Soltau, 496, 549, 886
Féré, 888
Fergusson, 886
Fiessinger, 5
Finkelstein, 748
Finlay, 665
Finny, 538
Fischer, 545
Fisher, T., 687, 715
Fitz, 879 *et seq.*
Fleiner, 208
Fontan, 776 *et seq.*, 955
Fort, 199
Foster, M., 324 (*note*), 464
Foster, W. B., 3, 221
Fothergill, 28 (*note*)
Foucart, 66
Fournier, 66 *et seq.*
Fowler, J. K., 801, 929
Fox, Wilson, 24, 433, 439, 450, 489
Foxwell, 637, 713
Francis, 298
Frank, J. P., 490
Fränkel, 608
Franks, W. Kendal, 369, 878
Frees, 670
Frerichs, 177, 207, 285, 550
Friedenwald, 558
Friedländer, 4
Friedleben, 130
Fuller, 234, 254
Fuller, W. H., 3, 19, 52
Furbringer, 431, 929

GABBETT, 4
Gaglio, 203
Galen, 195
Galliard, 517, 719
Gamgee, A., 223, 282 *et seq.*
Ganzinotty, 609
Gardner, 229
Garrod, A. B., 73, 155 *et seq.*
Garrod, A. E., 4 *et seq.*, 40, 84
Garrod, Alfred, 706
Gaston, 335
Gaucher, 200
Gee, S., 110, 968
Gemmel, 229
Gerhardt, C., 527
Gillespie, 20
Glaisher, 458
Gläser, 429
Glénard, 429, 480, 591
Gley, 229
Glisson, 108
Gmelin, 299
Goltz, 331
Goodeve, 776
Goodhart, 2 (*note*), 40, 463, 949
Gould, 547
Gowers, 68
Grant, 776
Grasset, 591
Grassius, 517
Graves, 474
Grawitz, 337
Greenhow, 20
Griffini, 525
Griffiths, J., 969
Grube, 217
Grünbaum, 329
Gübler, 350, 616
Guérin, 123
Gull, W. W., 11 (*note*), 217, 474
Gunsberg, 525
Günzburg, 289
Guttman, 929

HABERSHON, S. O., 439, 555, 640, 886, 939
Haeberlin, 538
Hager, 229 (*note*)
Haig, 3, 898
Hall, Marshall, 452
Hallervorden, 212
Hallier, 337
Hamburger, 362
Hamilton, D. J., 488
Hammarsten, 297
Hammond, 453
Hanot, 208, 721
Hansemann, 208
Harley, J., 883
Harley, Vaughan, 208
Harris, V. D., 372
Harrison, E., 212
Harrison, Reginald, 484
Hauser, 538
Haussmann, 719
Haward, 969
Hawkins, H. P., 637, 896 *et seq.*
Hayem, 28, 291, 509, 517, 723
Haygarth, 28 (*note*), 101
Head, H., 475, 528
Head, M., 557
Hebb, 680, 713
Hédon, 203
Hehner, 291
Heidenhain, 288, 464
Heineke, 547
Heitzman, 130
Henderson, 776
Henoch, 41, 641
Hepburn, 458 *et seq.*

Hermann, 464
Herringham, 967
Herying, 508
Hewitt, Graily, 450
Hildebrandt, 230
Hill, Berkeley, 800
Hill, Leonard, 331
Hillary, 776
Hillier, 47
Hippocrates, 155
Hirsch, 4
Hirschfeld, 220
Höck, 65
Hodenpyl, 891
Hoffmann, 204, 964
Holmes, T., 548, 805
Holst, 748
Holt, 960
Hope, J., 19
Hoppe-Seyler, 208, 296, 498 (*note*)
Horace, 76
Horowitz, 609
Howard, 454
Howse, 961
Hudson, 711
Hüfner, 298
Hugueneng, 220
Humboldt, 461
Humby, 492
Hunter, John, 324
Hunter, W., 300, 549
Hutchinson, J., 160, 336, 352, 835

IRWIN, 450

JACCOUD, 59, 106
Jacobi, 336
Jaksch, von, 213, 502
James, 450
Janeway, 927
Jaworski, 517
Jenner, W., 109, 432
Johnson, G. S., 214, 640
Jones, 432
Jones, Bence, 287
Jones, Handfield, 440
Jones, Lewis, 100
Jones, Sydney, 106
Jowers, 547

KAMMERER, 71
Kaposi, 217
Kassowitz, 112, 139
Kaufmann, 203
Kelsch, 776
Kelynack, 880, 895
Kennedy, 229
Kiener, 776
Kinch, 225
Kisch, 199
Kischenski, 671
Kjellberg, 753
Klebs, 439, 524
Klemperer, 288
Kocslakoff, 427
Koeberlé, 878
Konig, 670
Korczinski, 523
Krafft, 885
Kraus, 550
Krause, 245
Krismer, 25
Krusenstern, 296
Külz, 3, 216
Kundrat, 432, 492
Kunze, 20
Küssmaul, 221, 489 *et seq.*, 517
Kuster, 546
Küttner, 80 (*note*)

LABBÉ, 200
Laboulbène, 436
Lancereaux, 202, 519
Lane, Arbuthnot, 684
Lang, 519
Lange, 4
Langerhans, R., 525
Langstaff, G. B., 748
Lanz, Otto, 890
Lapersonne, 65
Latham, P. M., 775 (*note*)
Latham, P. W., 3, 205
Latham, R. M., 19
Lauro, 123
Lauwers, 674
Laves, 203
Law, 521
Lea, Sheridan, 284
Lebert, 19, 538, 673
Lees, D. B., 21, 53, 104 (*note*),
Legal, 215
Leichtenstern, 801 *et seq.*
Le Nobel, 215
Leo, 203, 291
Lépine, 203
Lesage, 303, 749
Letulle, 200, 517, 526, 714
Leube, 429, 470, 504, 517
Levison, F., 165
Lewis, 886
Leyden, 68, 537
Lieben, 215
Liebert, 525
Liebig, 201
Liebreich, 948
Light, E. M., 943
Lindemann, 65
Lindner, 670
Lingard, 339
Little, 970
Llobet, 679
Lockwood, C. B., 975
Long, 454
Longstaff, 3
Lorenz, 502

Lortet, 461
Louis, 640
Lubimoff, 207
Lucas, Clement, 65
Luff, 130, 767
Luschka, 486
Lustig, 209
Lyons, 6

MACALISTER, 480
MacCormac, William, 321
MacDonell, R. W., 69
Mackenna, 454
Mackenzie, H. W. G., 229
Mackenzie, M., 362 *et seq.*
Maclagan, J. T. 17, 57
Macleod, 19, 429
Macleod, Neil, 791
Makins, 711
Manges, 559
Manoury, 429
Mantle, 5
Marcet, 256, 458
Marchal, 218
Marfan, 749
Marie, 153
Marsh, Howard, 483
Marshall, 833
Martens, 508
Martin, 633, 776
Martin, Sidney, 473, 496, 518, 555, 660, 715
Mascarel, 429
Mathes, 527
Mathieu, 545
Matterstock, 885
Mayer, 439
Maylaro, 547
Mayo, 450
Mayow, Robert, 399
M'Naught, 556
Meckel, 637
Meding, 26
Melier, 910
Meltzing, 508
Menge, 609
Mering, von, 202, 291, 500
Meynet, 47
Middleton, 92
Miller, 492
Minkowski, 202
Mintz, 291, 429
Mitchell, J. K., 3
Moleschott, 229
Monckton, 229
Moncorvo, 102
Money, A., 968
Monin, 230
Moore, 776
Moore, Norman, 165, 555, 720
Morat, 205
Morehead, 26
Morisse, 608
Morton, 893
Moullin, Mansell, 325
Moxon, 13, 174, 525, 937
Mulder, 215
Müller, 502
Murchison, 974
Murphy, 885
Muskett, 126
Mylius, 298

NANCE, 726
Nasse, 439
Neuhaus, 450
Neuman, 520
Newman, D., 372
Newsholme, 4 *et seq.*
Niemeyer, 432
Noorden, von, 59, 203, 489
Normand, 777
Nothnagel, 754

OBRE, 799
Odling, 256
Ogle, C., 371
Oliver, T., 84, 183, 229, 318
Olshausen, 628
Ord, W. M., 70, 74
Ormerod, 956
Orth, 432, 440
Oser, 426, 492
Osler, 77, 506, 565, 659 *et seq.*, 681, 882, 970

PACANOWSKI, 506
Page, F., 487
Pagès, 296
Paget, James, 149
Paget, S., 968
Paijkull, 297
Parise, 804
Pariser, 481
Parkes, E. A., 234
Parrot, 127, 351, 432
Partridge, 805
Passet, 606
Pasteur, 59
Paton, Noel, 204, 296
Paviot, 442
Pavy, 201 256, 517
Pawlow, 427 464, 501
Payne, 91, 335
Payne, F. J., 645, 927
Peacock, 968
Peebles, 492
Peiper, 209
Penzoldt, 490
Pepper, 492
Percy, 207
Perry, 548, 554 *et seq.*, 713
Pertik, 709
Petrone, 71
Pettenkofer, 298

Peyrand, 229
Pfeiffer, 303, 750
Phillips, L. C. P., 7
Phillips, Sydney, 68
Piatkowski, 230
Pic, 720
Pilliet, 427
Pitcairn, David, 16
Pitres, 84
Pitt, G. N., 91, 363, 568, 714, 975
Planer, 964
Plaut, 337
Plowright, 184
Pollard, 710
Pollock, G. D., 832, 867
Poncet, 65
Ponfick, 549
Poore, Vivian, 13
Porter, 889
Potain, 520, 816
Power, D'A., 710
Priestley, J., 747
Prout, 3, 250
Pryce, Davies, 217
Puckham, 907

QUAIN, R., 833
Quincke, 208, 525

RAFINESQUE, 855 *et seq.*
Ralfe, 215
Ramond, 75
Ransom, W. B., 890
Rasmussen, 525
Rathery, 2 (*note*)
Raukie, 339
Rauzier, 154, 591
Rawlinson, 451
Rayher, 3
Raynaud, 219, 429
Recklinghausen, 439
Rees, Owen, 19, 337
Réfrégé, Félix, 872
Reichel, 604
Reichmann, 508, 534
Reignier, 809
Remy, 132
Renault, 538
Rendu, 200
Reynolds, E. S., 223
Ribbert, 885
Richardson, B. W., 3, 372
Riegel, 288, 485, 517, 527
Rindfleisch, 524
Ritter, 525
Roberts, F. T., 650
Roberts, William, 284
Robertson, 248
Robin, 337
Robinson, 957
Robson, Mayo, 296, 947, 966
Roger, 324
Rokitansky, 517, 590
Rolleston, 969
Rollet, 66
Rollo, 223
Rorsch, 670
Rosenbach, 450, 485
Rosenheim, 527
Ross, 528
Roth, 714
Roux, 927
Roy, 326, 457 *et seq.*
Rubner, 215
Rüdinger, 588
Ruge, 964
Russell, J. W., 214

SAMSON, JAMES, 633
Sanarelli, 286
Sandmeyer, 206
Sands, 859
Sansom, 74, 339
Sansoni, 203
Saundby, 243, 496
Savlief, 545
Schachmann, 208
Schäfer, 318
Schmitz, 200
Schnetter, 444
Scudamore, 157, 197
Sée, Germain, 229, 288
Seegen, 201
Seemann, 291
Seitz, 432
Selle, 64
Senator, 2, 41, 57, 91, 222, 502
Sevestre, 335, 749
Sharkey, 953
Shattock, 362, 710
Shaw, J., 841
Shaw, L. E., 548, 554 *et seq.*
Sherrington, 324
Shield, 676
Shüller, Max, 74
Sibson, 53
Silbermann, 526
Simpson, 947
Sjoquist, 291
Slater, C., 212
Smirnow, 432
Smith, Andrew, 459
Smith, J. W. F., 444
Smith, Pye, 721, 859
Smith, Shingleton, 206, 945
Sneller, 440
Snow, 128
Sohlern, von, 518
Sollier, 484
Southam, 636
Spender, Kent, 73 (*note*)
Spillmann, 609
Squire, Balmanno, 230
Stadelmann, 212
Starling, Ernest, 464
Steitzing, 545

Stengel, 492
Stevenson, 975
Stewart, Grainger, 429
Stich, 550
Straus, 559
Stricker, 427
Stromeyer, 20
Strong, Thomas, 867
Sturges, 16, 42
Suksdorff, 735
Sutton, Bland, 128, 676
Sutton, H. G., 11 (*note*), 499
Swediaur, 64
Sydenham, 1
Symington, 124
Symonds, C., 376

TAIT, LAWSON, 628
Talamon, 898
Tappeiner, 501
Tapret, 640
Targett, 371, 963
Tavel, 891
Taylor, F., 374, 659
Tenger, 895
Terillon, 603
Terrier, 800
Tessier, 234 *et seq.*
Testelin, 65
Thayer, 71
Thin, 776 *et seq.*
Thiroloix, 203
Thomas, Davies, 685
Thomson, A., 124
Thomson, John, 432
Thorburn, W., 153
Tizzoni, 316
Todd, 3, 76
Toepfer, 558
Tomkins, Henry, 747
Tootle, 950
Trastour, 969
Traube, 727
Travers, 322 (*note*)
Treves, F., 480, 567, 673
Tripier, 123
Trousseau, 108, 236, 476, 485
Twining, 776
Tyson, 196

UEFFELMANN, 290

VAILLARD, 84
Valentine, 229
Van der Burg, C. L., 776 *et seq.*
Van der Velden, 517
Vanni, 525
Vassale, 525
Vaughan, 735, 748
Vierordt, 640
Vincent, Georges, 721
Virchow, 257, 428, 517, 643, 889, 948
Voelcker, 372
Voit, 130, 204

WAGNER, 519
Waldeyer, 685
Walker, J. T., 515, 786, 969
Walters, Rufenacht, 153
Warner, F., 29, 47
Watson, T., 16, 45, 472
Weber, L., 7, 199
Wegele, 545
Wegner, 602
Weintraud, 203
Weir, 888
Welch, 517, 606
Wells, Spencer, 669, 970
Wertheim, 609
West, S., 15, 230, 636, 681
Wethered, 783
Weyl, 208
White, Hale, 226
Whittier, 722
Whittle, Glynn, 450
Whymper, 457
Wiggin, 876
Wilks, S., 229, 331, 380, 433, 525, 631 (*note*), 730, 889, 937
Williams, C. T., 976
Williams, Dawson, 747
Williams, Roger, 372
Williamson, R. T., 206
Willis, 196, 234
Wilson, A. H., 371
Winston, 296
Winter, 291
Wohlmann, 75
Wollaston, 155, 448, 450
Wollstein, 432
Wolpa, 216
Woodhead, Sims, 659
Wunderlich, 12

YEO, BURNEY, 898

ZABLUDOWSKI, 514
Zaleski, 208
Ziemssen, von, 504
Zweifel, 535

INDEX

ABDOMEN in intestinal obstruction, 842; in perityphlitis, 902
Abdominal diagnosis (gynæcological), 579; method of examination, 580; obscure cases, 583; uterine tumours of pregnancy, 581; not connected with pregnancy, 584
Abdominal neuralgias, various, 480
Abdominal wall in peritonitis, 614
Acetonuria, 222; in gastric ulcer, 532
Achondroplasia, 124
Acidity in dyspepsia, 392
Acromegaly, 151; bibliography, 153; diagnosis, 152; pathology, 152; symptoms, 151
Actinomycosis of the appendix in perityphlitis, 890, 917; of the small intestine, 719
Acute rheumatism, 1; age in, 10, 12; atmosphere and climatic conditions, influence of, 4; bibliography, 36; complications, 14, 22; diagnosis, 17; duration, 11; etiology and geographical distribution, 3; heredity in, 10; mortality, 1; onset, 11; relapses, 12; sequels, 28; sex in, 9; statistics, 2, 29 *et seq.*; symptoms, 11; treatment, 18
Acute rheumatism of childhood, 38; bibliography, 53; diagnosis, 40; prognosis, 51; sex in, 39; treatment, 52
Acute rickets, so-called, 123
Addison's disease, 319
Adenoma of the intestine, 830; of the stomach, 568
Adhesions, peritoneal, 645
Albuminuria and lardaceous disease, 270
Alcohol in gout, 182, 189
Alimentary canal in perityphlitis, 896; in sprue, 783
Amyloid, *see* Lardaceous disease, 255
Anæmia after rheumatic fever, 28; in acute rheumatism of childhood, 42
Anæmia, primary, diagnosis from ulcerative colitis, 954
Anazoturia, diagnosis from diabetes insipidus, 240
Anus and rectum, differential diagnosis of diseases of, 977
Angioma of the intestine, 830; of the peritoneum, 684
Anorexia nervosa, 474; treatment, 479
Aphthous stomatitis, 336
Appendicitis, *see* Perityphlitis, 879
"Appendicular colic," 910
Appendix in perityphlitis, 884; actinomycosis, 890, 917; catarrh, 884; cystic change, 889; gangrene, 887; perforation, 886; stricture, 888; ulceration, 885
Appetite in dyspepsia, 389
Aptyalism, 357
Arthritis deformans, *see* Rheumatoid arthritis, 73
Arthritis in acute rheumatism of childhood, 40; varieties of, 73
Asthma dyspepticum, 408
Atrophy of muscles in rheumatoid arthritis, 82
Azoturia, 250; causes, 252; treatment, 252

BACTERIAL necrosis in ulcer of stomach, 526
"Ballooning of the rectum," 838
Barlow's disease, *see* Scurvy rickets, 123
Baruria, 254
Baths in diabetes mellitus, 228; in rheumatoid arthritis, 99; in rheumatic fever, 26; in rickets, 139
Bile, 295; general characters of, 296
Bile acids, 298
Bile pigments, 299
"Biliousness" in childhood, 411
Black tongue, 351
Bladder in diabetes mellitus, 210
Blood in collapse, 326; in diabetes mellitus, 207; in gout, 162; in rickets, 122
Bones in rickets, 116
Bowels, in dilatation of the stomach, 498; in lardaceous disease, 263
Brain in diabetes mellitus, 206; in rickets, 122
Breath in dyspepsia, 389
Bright's disease, in connection with hæmorrhagic colitis, 962; with ulcerative colitis, 957
Bulimia in gastralgia, 472
Burns as cause of duodenal ulcer, 548

CANCER, *see* also Carcinoma
Cancer of the cæcum, diagnosis from perityphlitis, 925
Cancer of the colon, 827
Cancer of the stomach, diagnosis from gastric ulcer, 540
Cancrum oris, *see* Gangrenous stomatitis, 339
Capsulitis, 646
Carcinoma of the intestine, 827 ; of the stomach, 554
Carcinoma of the œsophagus, 371 ; age in, 372 ; diagnosis, 375 ; morbid anatomy, 373 ; prognosis, 376 ; situation of the growth, 372 ; symptoms, 375 ; treatment, 376
Carcinoma of the peritoneum, primary, 679 ; age in, 681 ; clinical characters, 682 ; diagnosis, 684 ; morbid anatomy, 681 ; physical signs, 683 ; prognosis, 684 ; secondary, 680 ; sex in, 681
Cardiac inflammations in acute rheumatism of childhood, 42
Cardiac lesions in gonorrhœal rheumatism, 69
Catarrhal stomatitis, 333
Catarrh of the appendix in perityphlitis, 884
Chlorosis, diagnosis from gastric ulcer, 539
Cholalic acid, 299
Cholera, diagnosis from intestinal obstruction, 872
Cholesterin, 296
Chondrodystrophia fœtalis, 124
Chorea in acute rheumatism of childhood, 51 ; in rheumatic fever, 23
Chronic fibrous rheumatism, 106
Chronic rheumatism, 56 ; bibliography, 60 ; in children, 58 ; sequels, 59 ; treatment, 60 ; types of, 56
Chyme, 286
Circulatory system in diabetes mellitus, 220 ; in gout, 160
Cirrhosis of the stomach, 440 ; bibliography, 444 ; etiology, 441 ; pathological anatomy, 440 ; symptoms, 443 ; treatment, 444
Climate in diabetes mellitus, 226 ; in lardaceous disease, 270, 276 ; in rheumatoid arthritis, 98 ; in rickets, 126
Colic, 727 ; bibliography, 732 ; causes, 729 ; description, 729 ; diagnosis, 730 ; diagnosis from peritonitis, 625
Colitis, 937 ; causes of, 948 ; "diphtheritic," 950 ; dyspeptic, 943 ; in connection with pneumonia, 949 ; with renal disease, 942, 949 ; membranous, 943 ; secondary, 947 ; simple, 937 ; ulcerative, 950
Colitis, acute, in connection with pneumonia, 942 ; diagnosis from perityphlitis, 923
Colitis, simple, 937 ; diagnosis, 940 ; prognosis, 940 ; symptoms, 938 ; treatment, 940 ; varieties, 941
Colitis, ulcerative, 950 ; age in, 950 ; associated diseases, 957 ; diagnosis, 954 ; etiology, 950 ; morbid anatomy, 955 ; prognosis, 953 ; sex in, 950 ; symptoms, 951 ; treatment, 958
Collapse, causes of, 322 ; accidental causes, 330 ; bibliography, 332 ; pathology, 325 ; treatment, 331
Collapse in intestinal obstruction, 839 ; in acute cases, 849
"Colloid cancer" of the peritoneum, 681
Colon, diseases of, 937 ; bibliography, 975 ; carcinoma of, 827 ; dilatation, 964 ; "idiopathic" dilatation, 967 ; laceration, 973 ; malformations, 975 ; metallic pigmentation, 975 ; perforation from without, 974 ; sacculation, 972 ; ulceration, follicular, 959 ; hæmorrhagic, 962 ; trophic, 963 ; vascular, 960
Coma in diabetes mellitus, 221, 231 ; in hydruria, 239
Congenital syphilis as a cause of rickets, 127
Constipation, 696 ; causes, 697 ; symptoms, 701 ; treatment, 702
Constipation in hydruria, 248 ; in intestinal obstruction, 841, 848 ; in peritonitis, 618, 653 ; in perityphlitis, 901
Corrosive poisons, action of, on stomach, 434
Cramps in intestinal obstruction, 849
Cranium in rickets, 113
"Cutaneous dystrophy" in rheumatoid arthritis, 82
Cysts in stricture of the appendix, 889

DEFECTIVE hygiene as a cause of rickets, 127
Deformities in rickets, treatment of, 141 ; in rheumatoid arthritis, 85
Diabetes insipidus, 234 ; age in, 237 ; bibliography, 255 ; classification, 234 ; diagnosis, 240 ; etiology, 235 ; nutrition, 239 ; pathology, 244 ; prognosis, 241 ; sex in, 235 ; symptoms, 237 ; treatment, 245 ; treatment, diabetic, 248 ; hygienic, 250
Diabetes mellitus, 195 ; age in, 198 ; bibliography, 231 ; complications, 217 ; diagnosis, 216 ; etiology, 196 ; exciting causes, 199 ; history, 195 ; morbid anatomy, 205 ; pathogenesis, 200 ; prognosis, 216 ; sex in, 198 ; statistics, 196 ; symptoms and course, 210 ; termination, 216 ; tests for sugar in urine, 213 ; treatment, 223 ; treatment of symptoms, 230
Diabetes mellitus and gout, 199
Diabetic gangrene, 218
Diaceturia in gastric ulcer, 532
Diaphragmatic hernia, 577 ; bibliography, 578
Diarrhœa, 733 ; bibliography, 745 ; causes, 733 ; forms, 733 ; symptoms, 737 ; treatment, 739
Diarrhœa in lardaceous disease, 273
Diarrhœa of children, 746 ; bibliography, 774 ; etiology, 746 ; morbid anatomy, 749 ; prevention, 759 ; treatment, 760 ; varieties, 749

Diarrhœa of children, chronic, 768; bibliography, 774; diagnosis, 772; etiology, 768; prognosis, 772; symptoms, 769; treatment, 772
Diarrhœa of children, inflammatory, 751; complications, 754; diagnosis, 755; duration, 754; prognosis, 756; symptoms, 751; treatment, 762
Diarrhœa of children, acute, 749; choleraic or septic, 757; chronic, 768; "dysenteric," 753; inflammatory, 751; "lienteric," 751; simple, non-inflammatory, 750
Diet in constipation, 703; in diabetes mellitus, 223; in diarrhœa of children, 762, 773; in dilatation of the stomach, 510; in gout, 187; in peritonitis, 626; in rheumatoid arthritis, 97; in rheumatic fever, 21; in rickets, 128, 134; in sprue, 788; in gastric ulcer, 542
Digestion, disorders of, in rickets, 128
Digestion, gastric, 286; intestinal, 295; oral, 281
Digestion, general pathology of, 281, 386; bibliography, 304
Digestive system in diabetes mellitus, 220
Dilatation of heart in acute rheumatism of childhood, 47; in rheumatic fever, 17
Dilatation of stomach, 485
Dropsy in acute rheumatism of childhood, 47
Drugs in rheumatoid arthritis, 100; in rickets, 138; in salivary secretion, 285
Duodenal pouches, 709
Duodenum, congenital occlusion of, 709; innocent tumours of, 713
Duodenum, malignant disease of, 720; diagnosis, 724; morbid anatomy, 720; situation of growth, 721; symptoms, 722; treatment, 725
Dyspepsia, 385; analysis of symptoms, 388; bibliography, 407; causation, 394; consequences, 400; symptoms, 387; treatment, 400
Dyspepsia, chronic, *see* Gastritis, chronic, 420
Dyspepsia in childhood, 407; acute gastric catarrh, 409; acute indigestion, 411; bibliography, 414; clinical varieties and symptoms, 410; intestinal dyspepsia, 413; gastric dyspepsia, 407; recurrent gastric catarrh, 412; subacute catarrh of the stomach, 411
Dyspepsia, nervous, 469

ECZEMA in diabetes, 217
Effusion in chronic peritonitis, 647
Electricity in hydruria, 248
"Encysted peritonitis," 612
Endocarditis in acute rheumatism of childhood, 42; in rheumatic fever, 15, 16
"Endothelial sarcoma" of the peritoneum, 680
Enteralgia, 481, 727; diagnosis from pelvic neuralgia, 482
Enteric fever and diabetes mellitus, 221; diagnosis from acute tuberculosis of the peritoneum, 666
Entero-colitis, *see* Diarrhœa of children, inflammatory, 751
Enteroliths in intestinal obstruction, 834
Enteroptosis, 587; anatomical considerations 587; bibliography, 597; displacements of individual viscera, 588; general ptosis of the abdominal viscera, 591; illustrative cases, 592; treatment, 596
Enthetic arthritis, 73
Epithelioma of the intestine, 827
Exophthalmic goitre, 319
Exudative erythemata in acute rheumatism of childhood, 50
Eye, affections of, in diabetes mellitus, 219; in gout, 160

"FACIES Hippocratica," 324
Fæcal accumulation in intestinal obstruction, 835, 866
Fæcal evacuation, physiology of, 693
Fæces, 302
Fasciæ in gonorrhœal rheumatism, 67
Feeding, *see* Diet
Fever in peritonitis, 619; in perityphlitis, 902
Fibro-lipoma of the peritoneum, 673; diagnosis, 675; physical signs, 674; prognosis, 675
Fibroma of the intestine, 830; of the peritoneum, 672; of the stomach, 568
Fibromyoma of the intestine, 830; of the peritoneum, 676; of the stomach, 568
"Fibrous rheumatism," 59
Flatulence in dyspepsia, 393; in gastralgia, 473
Fœtal cretinism, 124
Fœtal rickets, 123
Foreign bodies as cause of intestinal obstruction, 832, 857; of perityphlitis, 884, 898

GALL-STONE colic, diagnosis from perityphlitis, 922
Gall-stones, diagnosis from enteralgia, 482; from gastric ulcer, 540; from neuralgia, 476
Gangrene of the appendix in perityphlitis, 887; of the intussusceptum, 824; of the lung in gastric ulcer, 537
Gangrene of the stomach, 438; bibliography, 440; pathology, 438; symptoms, 440
Gangrenous stomatitis, 339
Gas, composition of, in carcinoma of the stomach, 556
Gastralgia, *see* Stomach, neuroses of, 469
Gastrectasis, *see* Stomach, dilatation of, 485
Gastric catarrh and dilatation of the stomach, 496; diagnosis from gastric ulcer, 539
Gastric digestion, 286
Gastric juice, 287; in ulcer of the stomach,

526 ; variations in chemical constitution of, 294
Gastric ulcer, *see* Stomach, ulcer of, 517
Gastritis, acute, 415 ; diagnosis, 418 ; etiology, 415 ; pathology, 418 ; symptoms, 416 ; treatment, 419
Gastritis as cause of ulcer of the stomach, 525
Gastritis, chronic, 420 ; bibliography, 423 ; diagnosis, 422 ; etiology, 420 ; pathology, 421 ; symptoms, 422 ; treatment, 423
Gastritis, membranous, 431 ; bibliography, 438
Gastritis, phlegmonous, 423 ; bibliography, 437 ; circumscribed form, 430 ; diffuse form, 424 ; etiology, 426 ; symptoms, 428
"Gastritis polyposa," 421
Geographical tongue, 350
Glanders, diagnosis from rheumatic fever, 18
Glossitis, 344 ; chronic, 345
Glycocholic acid, 298
Glycosuria, *see* Diabetes mellitus, 195
Gonorrhœal rheumatism, 64 ; bibliography, 72 ; clinical features, 66 ; diagnosis, 69 ; etiology, 65 ; pathology, 70 ; prognosis, 70 ; treatment, 71
Gout, 155 ; age in, 181 ; bibliography, 195 ; connection with other diseases, 184 ; diagnosis, 185 ; etiology, 181 ; general management, 187 ; immediate causes, 183 ; local treatment, 194 ; morbid anatomy, 163 ; pathology and chemistry, 166 ; predisposing causes, 182 ; premonitory signs, 157 ; prognosis, 185 ; recurrence of paroxysms, 156 ; retrocedent, 159 ; sex in, 181 ; symptoms and course of regular, 155 ; symptoms of irregular, 159 ; therapeutics, 186 ; treatment, 186
Gout, diagnosis from rheumatic fever, 18

HÆMATEMESIS in gastric carcinoma, 559 ; in gastric ulcer, 530 ; treatment of, 546
Hæmophilia, diagnosis from rheumatic fever, 18
Heart, in diabetes mellitus, 207 ; in rickets, 121
Hepatalgia, 481
Hepatic abscess, diagnosis from perityphlitis, 926
Heredity in diabetes mellitus, 198 ; in gastric ulcer, 520 ; in gout, 181 ; in acute rheumatism of childhood, 40 ; in rickets, 127
Hernia, diaphragmatic, 577 ; internal, 805
Hip disease, diagnosis from perityphlitis, 925
Hydrochloric acid in gastric carcinoma, 558
Hydruria, 235
Hyperacidity in gastric ulcer, 534
Hyperpyrexia in rheumatic fever, 23, 42 ; causation, 25 ; symptoms, 25 ; treatment, 26
Hypertrophic pulmonary osteo-arthropathy, 153 ; bibliography, 154 ; diagnosis, 154 ; pathology, 154 ; symptoms, 153
Hypertrophy of heart in acute rheumatism of childhood, 47
Hysteria, diagnosis from colic, 732

ILEO-CÆCAL valve, stricture of, 829
Infantile scurvy, 123 ; diagnosis from acute rheumatism, 41
Infantile palsy, diagnosis from rheumatic arthritis, 41
Injuries as cause of perityphlitis, 896
Innervation, disturbances of, in hydruria, 238
Intercostal rheumatism, 63
Internal hernias, 805 ; diaphragmatic, 806 ; intersigmoid, 807 ; into the foramen of Winslow, 807 ; into the fossa duodeno-jejunalis, 806 ; pericæcal, 808
Intestinal digestion, 295
Intestinal dyspepsia, primary, 300 ; secondary, 301
Intestinal obstruction, 794 ; age in, 797 ; bibliography, 879 ; differential diagnosis, 845 ; general diagnosis, 871 ; general observations, 794 ; morbid anatomy, 797 ; obstructing agents, 798 ; portion of bowel involved, 797 ; prognosis and terminations, 873 ; relative frequency of various forms, 796 ; sex in, 797 ; symptoms, 836 ; treatment, 874 ; varieties, 796
Intestinal obstruction, acute form, 837, 846, 874 ; by acute kinking due to traction, 809 ; by adhesions which compress the gut, 812 ; by adhesions which retain bowel in a bent position, 810 ; by fæcal accumulation, 835 ; by foreign substances within the bowel, 832, 857 ; by gall-stones, 834, 857, 876 ; by intestinal concretions, 834 ; by intussusception, 819 ; by matting together of intestinal coils, 812 ; by narrowing after inflammation, 814 ; by pressure of tumours and other diseases external to the bowels, 835 ; by strangulation by bands or through apertures, 797, 850 ; by strangulation over a band, 808 ; by stricture, 825 ; by tumours, 830 ; by volvules, 815 ; chronic form, 837, 860, 876 ; chronic form ending acutely, 839, 870 ; in small intestine compared with obstruction in large, 843
Intestinal obstruction, diagnosis from peritonitis, 625 ; from perityphlitis, 923 ; from ulcerative colitis, 954
Intestinal strangulation, 797 ; anomalous forms, 808 ; by a peritoneal false ligament, 798 ; modes of, 800 ; by Meckel's diverticulum, 802 ; modes of, 804 ; by normal structures abnormally attached, 804 ; by omental cords, 801 ; through slits and apertures including internal hernias, 805
Intestine, diminution of expulsive power in,

697 ; displacement of, 590 ; increased resistance in, 699 ; movements of, 693 ; natural stimulants of, 698 ; nervous connections of, 694
Intestines, *see also* Colon, Small intestine
Intestines in diabetes mellitus, 209
Intussusception, 819 ; acute form, 852, 875 ; anatomical varieties, 819 ; causes, 824 ; chronic, 868, 876, 878 ; how obstruction, strangulation, and irreducibility are produced, 822 ; irreducibility of, 822 ; mesentery in, 822 ; mode of growth, 820 ; pathological changes in, 822 ; relative frequency of various forms, 820 ; retrograde, double and triple, 821
Intussusception of the dying, 821
Irritants, action of, on stomach, 435

JEJUNUM, and ileum, *see* Small intestine, innocent tumours of, 713 ; primary malignant disease of, 725
Joints in gonorrhœal rheumatism, 66 ; in gout, 157, 164 ; in rheumatic fever, 13

KIDNEYS in diabetes mellitus, 209 ; in gout, 166 ; in lardaceous disease, 259

LACTIC acid in gastric carcinoma, 558
Lardaceous disease, 255 ; age in, 271 ; bibliography, 277 ; chemistry, 255 ; etiology, 265 ; general pathology, 255 ; pathological anatomy, 259 ; sex in, 272 ; subjects of, 271 ; symptoms, 272 ; tables of causes, 266 ; table of chief causes of death, 275 ; treatment, 275
Laryngismus stridulus in rickets, 111 ; treatment, 142
Late rickets, 124
Lavage in dilatation of the stomach, 511
Lead poisoning and gout, 183
Lecithin, 297
Leontiasis ossea, 150
Leukoplakia, 346
Ligaments in rickets, 120
Lipoma of the intestine, 830 ; of the peritoneum, 673
Lips, cracking of, eczematous, 343 ; syphilitic, 342
Liver, displacements of, 589
Liver in diabetes mellitus, 204, 208 ; in lardaceous disease, 257, 261 ; in rickets, 121
Liver, tropical abscess of, in connection with ulcerative colitis, 957
Lumbago, 63
Lungs in diabetes mellitus 208 ; in rickets, 121
Lymphadenoma of the small intestine, 714 ; of the stomach, 568
Lymphatic glands in lardaceous disease, 264 ; in rickets, 122
Lympho-sarcoma of the intestine, 832

MALARIA and lardaceous disease, 270
Malignant disease of stomach, diagnosis from gastralgia, 477
Massage in dilatation of the stomach 514 ; in rickets, 139
Meckel's diverticulum, 710
Melæna in duodenal ulcer, 551 ; in gastric ulcer, 531
Mesenteric glands in tubercle of the peritoneum, 659
Mesentery in intussusception, 822
Metastatic parotitis, 358
Meteorism in intestinal obstruction, 842, 850 ; in peritonitis, 618
Micro-organisms of peritonitis, 605 ; of the mouth, 286
Miliaria in rheumatic fever, 14
Mollities ossium, *see* Osteomalacia, 147
Motor diseases of the stomach, 475
Mountain-sickness, 456 ; bibliography, 462 ; causation, 456 ; symptoms, 460
Mouth, diseases of, 333 ; bibliography, 359 ; micro-organisms of, 286
Mouth in sprue, 780, 789
Movable kidney, diagnosis from gastric ulcer, 541
Mucin in saliva, 284
Mucoid nucleo-albumin, 297
Mucous membranes in rickets, 121
Muscles in gonorrhœal rheumatism, 67 ; in rickets, 120
Muscular rheumatism, 61 ; bibliography, 64 ; treatment, 63
Myalgia, *see* Muscular rheumatism, 61
Myocarditis in acute rheumatism of childhood, 47 ; in rheumatic fever, 17
Myoma of the peritoneum, 676

NEPHRALGIA, 483
Nerves in diabetes mellitus, 207, 218 ; in hydruria, 244
Nervous disorders in rickets, treatment of, 142
Nervous dyspepsia, diagnosis from gastric ulcer, 540
Nervous influences in dyspepsia, 399
Nervous structures in gonorrhœal rheumatism, 68
Nervous symptoms in rickets, 110 ; treatment, 142
Nervous system in gout, 160
Neural arthritis, 73
Neuralgia, abdominal, diagnosis from colic, 732
Noma, *see* Gangrenous stomatitis, 339

OBESITY in diabetes mellitus, 199
Ocular lesions in gonorrhœal rheumatism, 68
Œsophageal pouches, 363
Œsophagismus, 378
Œsophagitis, 366 ; chronic, 368 ; diphtheritic, 366 ; pseudo-membranous, 366 ; simple acute, 366 ; traumatic, 367

Œsophagus, diseases of, 360; anatomy, 360; bibliography, 381; congenital malformations, 362; dilatation of, 365; innocent tumours, 377; methods of examining, 361; neuroses of, 378; pericœsophageal abscess, 377; phlegmonous inflammation, 367; rupture, 380; simple ulceration, 379; stricture, 369; stricture, fibrous, 369; malignant, 371; spasmodic, 378; varicose veins, 380
Omental tumour in tubercle of the peritoneum, 657, 663
Oral digestion, 281, 304
Oral mucous membrane, symptomatic appearances of, 342
Osteitis deformans, 149; bibliography, 150; pathology, 149
Osteomalacia, 147

PAIN in acute intussusception, 852; in carcinoma of the stomach, 557; in duodenal ulcer, 550; in intestinal obstruction, 839, 846; in peritonitis, 614; in perityphlitis, 900; in rheumatoid arthritis, 90; treatment of, 101; in ulcer of the stomach, 527; treatment, 546
Pancreas, 300; in diabetes mellitus, 202, 208
Papillomata of the stomach, 568
Papillomatosis diabetica, 217
"Paralytic secretions," 307
Parasitic stomatitis, 337
Pelvis in rickets, 116
Pemphigus vegetans, 336
Pepsin, 287, 295
Perforation in carcinoma of the stomach, 562; in duodenal ulcer, 551; of the appendix in perityphlitis, 886; of the stomach, diagnosis from colic, 731
Perforation in ulcer of the stomach, 522, 535; localised abscess as result, 537; treatment, 546; diagnosis from perityphlitis, 922
Pericardial fibrosis in acute rheumatism of childhood, 45
Pericarditis in acute rheumatism of childhood, 44; in rheumatic fever, 15, 25
Perinephritic abscess, diagnosis from perityphlitis, 925
Pericœsophageal abscess, 377
Peritoneal cysts, 685; clinical characters, 687; dermoid, 686; diagnosis, 688; duration, 688; hydatid, 685; of doubtful nature, 686; "sanguineous," 687
Peritoneum, new growths of, 672; angioma, 684; bibliography, 689; carcinoma, 679; "colloid cancer," 681; "endothelial sarcoma," 680; fibro-lipoma, 673; fibroma, 672; fibro-myoma, 676; lipoma, 673; myoma, 676; sarcoma, 676
Peritoneum, tubercle of, 654; acute form, 661; bibliography, 671; chronic, 662; course, signs and symptoms, 661; diagnosis, 666; fibrous, 656, 66
555; morbid anatomy, 654;
658 prognosis, 668; stati
treatment, 668; ulcerous, 656
"Peritonism," 616
Peritonitis, acute primary, 601; a
an ordinary case, 613; bibliog
diagnosis, 624; due to inf
intestine, 605; due to infe
without, 607; due to rheuma
due to the gonococcus, 609; et
general condition of patient, 6
considerations, 601; morbid
611; of doubtful nature, 608
measures, 630; pneumococcal
gnosis, 623; sex in, 610; sym
table of cases, 610; treatment,
culous, 608; varieties, 604, 6
Peritonitis, acute secondary, 631
with erysipelas, 631; bibliog
epidemic, 632; idiopathic, 636
633; symptoms, 634; with eru
632; with nephritis, 635
Peritonitis, chronic, 638; age
bibliography, 654; chronic con
prolonged ascites, 642; diag
etiology, 638; extension fro
structures, 643; idiopathic, 6
nant, 644; morbid anatomy, 64
signs, 649; prognosis, 652; s
symptoms, 650; toxic, 641;
643; treatment, 652
Peritonitis deformans, 645
Peritonitis, diagnosis from intesti
tion, 872; from perityphliti
children, 623; in perityph
masked, 623; of the new-
simple, diagnosis from tuberc
tuberculous, *see* Peritoneum,
654
Perityphlitic abscess, 892
Perityphlitis, 879; age in, 8
graphy, 937; classification, 899
tions, 926; constitutional con
905; diagnosis, 921; etiol
historical account, 879; morb
and pathology, 881; morts
normal anatomy, 880; oper
ference, 932; relative frequency
899; sex in, 895; symptoms,
ment between attacks, 931;
during attack, 929
Perityphlitis, actinomycotic, 9
915; chronic cases, 913; ma
913; mildest type, 909; most
911, 932; ordinary, ending in
906; relapsing, 918, 935; sp
912; taking origin from app
taking origin from cæcum,
abscess in, 892; the peritoni
tuberculous, 916
Perspiration in rheumatic fev
rickets, 110

Pharyngoceles, 363
Phosphaturia, 253 ; treatment, 254
Pigmentation of skin in rheumatoid arthritis, 87
Pleurisy in tubercle of the peritoneum, 660
Podagra, *see* Gout, 155
Polypi of the intestine, 713, 831
Polyuria, 250
Prolapsus ani in diarrhœa of children, 765
Prostration in diarrhœa of children, 765
Pruritus in diabetes mellitus, 230
"Pseudo-ileus," 628
Psilosis, *see* Sprue, 774
Psoas abscess, diagnosis from perityphlitis, 926
Pulmonary disorders in rickets, treatment of, 142
Purpura rheumatica in acute rheumatism of childhood, 50
Pyæmia in rheumatic fever, 18
Pylephlebitis, diagnosis from perityphlitis, 926
Pyroses in dyspepsia, 393

RACE, influence on rheumatic fever, 5
Renal colic, diagnosis from perityphlitis, 922
Renal inadequacy, diagnosis from diabetes insipidus, 240
Rennin, 287, 295
Respiratory organs in gout, 161 ; in diabetes mellitus, 219
Rheumatic fever, *see* Acute rheumatism, 1
Rheumatic series, the, in acute rheumatism of childhood, 38
Rheumatic torticollis, 63
Rheumatism, acute, 1 ; chronic, 56 ; gonorrhœal, 64 ; intercostal, 63 ; muscular, 61
Rheumatism in diabetes mellitus, 221
Rheumatoid arthritis, 73 ; bacteriology, 74 ; bibliography, 102 ; cautions and safeguards, 93 ; Charcot's classification, 76 ; clinical history, 80 ; definition, 73 ; in childhood, 51, 91 ; modes of beginning, 81 ; morbid anatomy, 79 ; nervous origin of, 83 ; pathology, 73 ; primary form, 74 ; secondary form, 75 ; treatment, 96
Rheumatoid arthritis in children, 91, 102, 106 ; bibliography, 107 ; diagnosis, 105 ; morbid anatomy, 105 ; symptoms, 103 ; treatment, 105 ; varieties, 102
Rickets, 108 ; bibliography, 143 ; diagnosis, 133 ; etiology, 125 ; general pathology, 132 ; histological changes, 119 ; history, 108 ; morbid anatomy, 112 ; prognosis, 134 ; short description, 108 ; symptoms, 109 ; treatment, curative, 136 ; of concurrent disorders, 141 ; preventive, 134
"Rickety osteomalacia," 124

"SAGO spleen," 263
Saliva, 281 ; action of drugs on, 285 ; activity of ptyalin, 283 ; alteration of reaction, 282 ; alterations in quantity, 283 ; alterations of ptyalin, 284 ; composition of, 281 ; diminished secretion of, 357 ; hypersecretion, 357 ; influence of disease on, 286
Salivary glands, affections of, 357 ; bibliography, 359 ; inflammation of, 358
Salts in bile, 299 ; in saliva, 285
Sarcoma of the intestine, 831 ; of the small intestine, 725 ; of the stomach, 567
Sarcoma of the peritoneum, 676 ; diagnosis, 678 ; prognosis, 679 ; signs and symptoms, 677
Scarlatinal rheumatism in children, 51
Scurvy rickets, 123 ; diagnosis from rheumatic fever, 18
Sea-sickness, 445 ; bibliography, 455 ; complications, 449 ; course and progress, 448 ; etiology, 445 ; pathology, 450 ; symptoms, 446 ; treatment, prophylactic, 453 ; remedial, 454
Seasons, influence on rheumatic fever, 7
Secretion, general pathology of, 304 ; bibliography, 320
Secretions, external, 306 ; of calculi, 311 ; of sweat, 310 ; of toxic substances, 313 ; of urine, 311 ; pathology, 310 ; uses of, 309
Secretions, internal, 313 ; bibliography, 320 ; by kidneys, 317 ; by liver, 317 ; by pancreas, 317 ; by suprarenals, 316 ; by thyroid, 314 ; pathology, 318
Senses, affections of, in diabetes mellitus, 219
Serous membranes in gonorrhœal rheumatism, 69
Shock and collapse, 320
Shock, causes of, 321 ; bibliography, 332 ; pathology, 323 ; prevention, 330 ; symptoms, 322 ; treatment, 330
Shock in peritonitis, 616
Sigmoid in dilatation of the colon, 968
Skin, affections of, in gout, 160, 194
Skin in diabetes mellitus, 217 ; in rheumatic fever, 11, 13 ; in rickets, 120
Sleeplessness in rheumatic fever, 13
Small intestine, diseases of, 709 ; bibliography, 727 ; congenital occlusion of, 712 ; malformations, 709 ; multiple diverticula, 711 ; primary malignant disease, 719 ; secondary growths, 726 ; ulceration, 715 ; vascular disturbances, 714
Spinal cord in diabetes mellitus, 206
Spinal disease, diagnosis from rheumatic fever, 18
Spine in rickets, 116
Spleen, displacements of, 589
Spleen in diabetes mellitus, 208 ; in lardaceous disease, 257, 263 ; in rickets, 122
Sprue, 774 ; bibliography, 794 ; etiology, 777 ; geographical distribution, 776 ; literature, 776 ; nomenclature, 775 ; path-

ology and pathological anatomy, 782; symptoms, 778; treatment, 786; types, 779
Starvation, effect on tissues, 782
Stenoses of large intestine, 843, 863; of small intestine, 712, 843, 860, 865
Stercoral ulcer in intestinal obstruction, 836; in perityphlitis, 882
Stomach, carcinoma of, 554; diagnosis from gastric ulcer, 564; cirrhosis of, 440
Stomach, contents of, chemical examination of, 288; physiology of, 293; qualitative tests for the various free acids, 289; quantitative estimation of acids, 290; soluble products of, 292; tests for gastric ferments, 292
Stomach, dilatation of, 485; acute form, 492; bibliography, 516; causation, 487; chronic, 493; diagnosis, 508; morbid anatomy, 507; nervous symptoms, 501; physical signs, 502; prognosis, 510; symptoms, 497; torsion in, 497; treatment, 510
Stomach, displacements of, 588; gases of, 293; micro-organisms of, 292; motor disorders of, 475; secondary growths in, 568; tumours of, 554, 568
Stomach in diabetes mellitus, 209; in lardaceous disease, 263; in tight lacing, 500
Stomach, gangrene of, 438
Stomach, neuroses of, 462; bibliography, 484; causes, 468; diagnosis, 476; introduction, 462; symptoms, 469; symptoms other than gastric, 476; treatment, 478
Stomach, ulcer of, 517; acute and chronic, 521; age in, 518; associated diseases, 519; causes, 524; complications and sequels, 535; course and duration, 538; diagnosis, 539; etiology, 517; forms of, 539; functional manifestations of, 534; habits, 519; heredity in, 520; history, 517; occupation, 519; pathogenesis, 523; pathological anatomy, 520; perforation, 522; prognosis, 541; race and climate, 518; sex in, 518; symptomatology, 527; traumatism, 520; treatment, 542
Stomatitis, aphthous, 336; gangrenous, 339; ulcerative, 334
Stools in chronic diarrhœa of children, 770; in sprue, 785, 789
Strangury in intestinal obstruction, 849
Stricture of the appendix in perityphlitis, 888; of the intestine, 712, 825; cancerous, 827; cicatricial, 825; condition in relation to clinical aspect of case, 828; position and relative frequency of different forms, 829; of the œsophagus, 369
Subcutaneous tendinous nodules in acute rheumatism of childhood, 29, 46
Subphrenic abscess, 537, 570; bibliography, 575; etiology and symptoms, 570; treatment, 575
Sudamina in rheumatic fever, 14
Suppuration as cause of lardaceou 265
Suprarenal capsules in lardaceou 264
Syphilis, as cause of lardaceous dis diagnosis from ulcerative colitis,
Syphilitic epiphyseal disease, diag rheumatic arthritis, 41
Syphilitic rickets, 123
Syphilitic ulceration of intestines,
Syphonage in dilatation of the ston

TABES mesenterica, *see* Peritoneum of, 659
Taurocholic acid, 299
Temperature in diabetes mellitus, hydruria, 239; in rheumatic 23; in acute rheumatism of c 42
Tenesmus in intestinal obstruction acute intussusception, 855
Tetany in dilatation of the stoma in rickets, 111; treatment of, 1
Thorax in rickets, 114
Thrombosis in gout, 172
Thrush, 337, 366
Thyroidectomy, 314
Thyroiodin, 315
Tongue, diseases of, 344; acute 346; actinomycosis, 357; appe health, 352; bibliography, 359 trophy, 356; inflammation innocent tumours, 357; leukopla lupus, 349; nervous affectio ulcerations, 348; wandering ras
Toxic gastritis, 433; bibliograp pathological anatomy, 433; s 436; treatment, 437
Tubercle of the stomach, 567
Tuberculous ulceration of the i 715; anatomical characters, 7 gnosis, 718; modes of attack, 71 tion, 716; symptoms, 716; t 718
Tumour in intestinal obstruction, 8
Tumours of the stomach, 554; bibl 569; complications, 561; det 560; diagnosis, 564; etiology, tology, 563; pathology, 562; 566; surgical treatment, 566; s 555; termination, 562; treatme
Tympanites, 964; causes, 965; s 971; treatment, 965
Typhoid fever, diagnosis from per 923
"Tyrotoxicon," 735

ULCER of duodenum, 547; age bibliography, 553; diagnosis, gnosis from gastric ulcer, 540; 548; pathological anatomy, 549

548 ; symptomatology, 550 ; treatment, 553
Ulcer of stomach, *see* Stomach, ulcer of, 517
Ulceration of the appendix in perityphlitis, 885 ; of the cæcum in perityphlitis, 882
Ulceration of colon, 959
Ulcerative endocarditis in acute rheumatism of childhood, 44
Ulcerative stomatitis, 334
Ulcers, intestinal (*see* Colon, Small intestine), causing cicatricial stricture, 826 ; in sprue, 785
Umbilical fæcal fistula in tubercle of peritoneum, 657, 664, 667
Uræmia, diarrhœa in, 735
Uratæmia in gout, 166
Uratic deposits in gout, 158 ; abarticular, 165 ; articular, 164 ; influence of saline substances, 173 ; influence of sodium salts, 174 ; influence of synovia, 175 ; mode of injury, 178 ; precipitation of, 171 ; resolution, 177 ; topography, 173
Uratosis in gout, 166 ; chemistry, 167 ; experiments in, 169-176
Uric acid gravel in gout, 162, 181
Urine in diabetes mellitus, 212 ; in dilatation of the stomach, 498 ; in gout, 161 ; in hydruria, 237 ; in intestinal obstruction, 843 ; in acute cases of, 849 ; in rheumatic fever, 11, 14 ; in rickets, 122

VILLOUS tumours of the stomach, 568
Volvulus, 815 ; of the ascending colon and cæcum, 817 ; of the sigmoid flexure, 815 ; of the small intestine, 818 ; symptoms, 851 ; treatment, 875
Vomiting, in acute intussusception, 853 ; in chronic, 869 ; in carcinoma of the stomach, 558 ; in diarrhœa of children, 766 ; in dilatation of stomach, 499 ; in dyspepsia, 390 ; in gastralgia, 472, 479 ; in gastritis, 429 ; in intestinal obstruction, 840, 864 ; in acute cases of, 846 ; in peritonitis, 616 ; in perityphlitis, 901 ; in ulcer of the stomach, 529, 546

WANDERING spleen, 589
Weak digestion in childhood, 407

XANTHOMA diabeticorum, 217
Xerostomia, 357

END OF VOL. III

Works on Medical Science and Cognate Subjects.

PUBLISHED BY

THE MACMILLAN COMPANY.

ANDERSON (J. W.). — **Lectures on Medical Nursing.** 16mo. 75 cents.
Essentials of Physical Diagnosis of the Chest and Abdomen. 75 cents.

"Dr. Anderson has written in a style as compact as possible, with a view to the economy of time, yet has satisfactorily covered his subject." — *N. Y. Times.*

BALFOUR. — **The Senile Heart.** Its Symptoms, Sequelæ, and Treatment. By GEORGE WILLIAM BALFOUR, M.D. 12mo. Cloth. $1.50.

"Cleverly written in a thoroughly interesting and practical style." — *American Medico-Surgical Bulletin.*

"A very clearly expressed and readable treatise upon a subject of great interest and importance. We can heartily commend this work as a valuable contribution to the literature of old age." — *Medical Record.*

BALLANCE (C. A.) and **EDMUNDS** (W.). — **A Treatise on the Ligation of the Great Arteries in Continuity.** With Illustrations. Royal 8vo. $4.00.

"This book will become a classic. It is on a timely and important subject, and is the fruit of deep and thorough research." — *Medical and Surgical Journal.*

BARWELL (R.). — **Lateral Curvature of the Spine.** $1.75.

BICKERTON (T. H.). — **On Colour-blindness.** Illustrated. *Nature Series.* (*In the press.*)

BLYTH (A. W.). — **A Manual of Public Health.** 8vo. $5.25.
Lectures on Sanitary Law. 8vo. $2.50.

BRODIE. — **Dissections Illustrated.** A Graphic Handbook for Students of Human Anatomy. By C. GORDON BRODIE, F.R.C.S., Senior Demonstrator of Anatomy, Middlesex Hospital Medical School; Assistant Surgeon, Northwest London Hospital. With plates drawn and lithographed by PERCY HIGHLEY. Complete in one volume. $9.00.

Part I. The Upper Limb. With seventeen Coloured Plates, two-thirds natural size, and ten Diagrams. $2.00.

"The plates are exceedingly well drawn and placed on the stone. . . . The explanatory letterpress is clear and concise."—*The Lancet.*

"Of such high merit are the plates of this, the first volume, that the publication of the three remaining numbers of the series will be awaited with impatience by all students of anatomy."—*The Hospital.*

"The scheme is admirably carried out and the plates most reliable. . . . We can confidently recommend the work."—*Guy's Hospital Gazette.*

Part II. The Lower Limb. With twenty Coloured Plates and six Diagrams. $2.50.

Part III. Head, Neck, and Thorax. With 20 Plates. $2.50.

Part IV. Abdomen. With 16 Plates, 13 Diagrams. $2.50.

BRUNTON.—Works by T. LAUDER BRUNTON, M.D., F.R.S.

On Disorders of Digestion: their Consequences and Treatment. 8vo. $2.50.

Pharmacology and Therapeutics; or, Medicine Past and Present. $1.50.

An Introduction to Modern Therapeutics. 8vo. $1.50.

BURDON-SANDERSON (J.).—**Memoirs on the Physiology of Nerve, of Muscle, and of the Electrical Organ.** Edited by J. BURDON-SANDERSON, M.D., F.R.SS.L. & E. 8vo. $5.25.

CARRINGTON (R. E.).—**A Manual of Dissections of the Human Body.** Second Edition, revised and enlarged. $2.50.

CARTER (R. B.).—**Eyesight:** Good and Bad. Second Edition, enlarged. With Illustrations. 8vo. $1.00.

CREIGHTON.—**A History of Epidemics in Great Britain.** By CHARLES CREIGHTON, M.A., M.D., formerly Demonstrator of Anatomy in the University of Cambridge. 8vo.

Vol. I. From A.D. 664 to the Extinction of the Plague. $4.50.

Vol. II. From the Extinction of the Plague to the Present Time. 8vo. $5.00.

CROCKER.—**Atlas of the Diseases of the Skin.** In a Series of Illustrations (coloured) from Original Drawings, with Descriptive Letterpress. By H. RADCLIFFE CROCKER, M.D., F.R.C.P., Physician, Skin Department, University College Hospital, London; formerly Physician to the East London Hospital for Children; Examiner in Medicine, Apothecaries' Hall, London. Superbly illustrated in colours. The plates (96 in number) represent fully 200 life-size figures, and are reproduced by chromo-lithography from the original water-colour drawings. Each fasciculus consists of six full-page plates, with descriptive letterpress. The letterpress not only describes the cases from which the drawings are taken, but also gives a general account of each disease, its differential diagnosis, and treatment. To be issued in 16 folio bi-monthly parts. Each part, $6.00. *Sold only on subscription to the entire set.*

DOLAN (T. M.).—**Pasteur and Rabies.** By THOMAS M. DOLAN, M.D., F.R.C.S. 16mo. 50 cents.

DOWNIE.—**Clinical Manual for the Study of Diseases of the Throat.** By JAMES WALKER DOWNIE, M.B. $2.50.

ECCLES. — Sciatica. A Record of Clinical Observations on the Causes, Nature, and Treatment of Sixty-eight Cases. By A. SYMONS ECCLES. $1.00.

"The generally accepted views touching the origin, symptoms, and treatment of sciatica, are lucidly presented in this brief record of personal observations. The author does not aim to furnish an elaborate monograph on the subject. As a clinical contribution to sciatica the little book will be read with interest." — *Medical Record.*

The Practice of Massage: Its Physiological Effects and Therapeutic Uses. By A. SYMONS ECCLES, M.B. Aberd., Member Royal College Surgeons, England; Fellow Royal Medical and Chirurgical Society, London; Member Neurological Society, London; etc. 8vo. $2.50.

FAYRER. — On the Preservation of Health in India. By Sir J. FAYRER, K.C.S.I., M.D., F.R.S. 18mo. 35 cents.

FEARNLEY (W.). — **A Course of Elementary Practical Histology.** $2.00.

FITZGERALD (C. E.). — **Lectures on Physiology, Hygiene, etc.** With Illustrations and Diagrams. 75 cents.

FLÜCKIGER (F. A.) and **HANBURY** (D.). — **Pharmacographia.** Second Edition, revised. 8vo. $5.00.

FOSTER. — A Text-Book of Physiology. By MICHAEL FOSTER, M.A., M.D., LL.D., F.R.S., Professor of Physiology in the University of Cambridge, and Fellow of Trinity College, Cambridge. 8vo. With Illustrations. Sixth Edition, largely revised.

Part I. Blood; the Tissues of Movement; the Vascular Mechanism. $2.60.

Part II. The Tissues of Chemical Action; Nutrition. $2.60. (*In the press.*)

Part III. The Central Nervous System. $1.75.

Part IV. The Central Nervous System (*concluded*); **The Tissues and Mechanisms of Reproduction.** $2.00.

Part V. (Appendix) **The Chemical Basis of the Animal Body.** By A. SHERIDAN LEA, M.A., Sc.D., F.R.S. $1.75.

"The present edition is more than largely revised. Much of it is re-written, and it is brought quite abreast with the latest wave of progress of physiological science. A chief merit of this work is its judicial temper, a strict sifting of fact from fiction, the discouragement of conclusions based on inadequate data, and small liking shown toward fanciful though fascinating hypotheses, and the avowal that to many questions, and some of foremost interest and moment, no satisfying answers can yet be given." — *New England Medical Journal.*

"It is in all respects an ideal text-book. It is only the physiologist, who has devoted time to the study of some branch of the great science, who can read between the lines of this wonderfully generalized account, and can see upon what an intimate and extensive knowledge these generalizations are founded. It is only the teacher who can appreciate the judicial balancing of evidence and the power of presenting the conclusions in such clear and lucid forms. But by every one the rare modesty of the author in keeping the element of self so entirely in the background must be appreciated. Reviewing this volume as a whole, we are justified in saying that it is the only thoroughly good text-book of physiology in the English language, and that it is probably the best text-book in any language." — *Edinburgh Medical Journal.*

"From its first issue as a single octavo volume of moderate size, in 1876, it has so grown that each of the five Parts is, in this sixth edition, nearly as large as the entire original work. From the beginning it was regarded as a masterpiece, and at once took a prominent place among text-books of physiology. . . . If one seeks for the reason of the high estimate in which this work is held on both sides of the Atlantic, by the most advanced students as well as by general readers, it may be found in the beauty and simplicity of the style, in the lack of personal prejudice on the part of the author, in

his thorough familiarity with the progress of physiological knowledge, and in the rare judgment with which purely hypothetical ideas and those founded on sufficient evidence are discriminated. The work is therefore a most admirable guide to physiological progress as well as general physiological knowledge." —*The Nation.*

FOSTER.—Text-Book of Physiology. In one volume. By MICHAEL FOSTER, M.A., M.D., etc., etc. Abridged and revised from the Sixth Edition of the Author's larger Work published in five octavo volumes. 8vo. Cloth, $5.00; Sheep, $5.50.

This new Edition will contain all the Illustrations included in the larger work, and will be published in one octavo volume of about 1000 pages. It will contain all of the author's more important additions to the complete work, and be like the sixth edition of that copyrighted in this country.

FOSTER (M.) and **BALFOUR** (F. M.).—**Practical Embryology.** With Illustrations. $2.60.

"A book especially adapted to the needs of medical students, who will find in it all that is most essential for them to know in the elements of vertebrate embryology."—*Academy.*

FOSTER (M.) and **LANGLEY** (J. N.).—**A Course of Elementary Practical Physiology.** Fifth Edition, enlarged. $2.00.

"This work will prove of great value to the teacher of physiology, as an aid to the preparation of an eminently practical course of lectures and demonstrations of elementary experimental physiology. Its chief utility, however, will be to the intelligent student, who, armed with a dissecting case, a microscope, and the book, will be enabled to pass his summer vacation in a manner at once interesting and profitable."—*Medical Record.*

FOSTER (M.) and **SHORE** (L. E.).—**Elementary Physiology.** 16mo. Illustrated. 75 cents.

"It is a veritable *multum in parvo*, and will be fully appreciated by those for whom it is intended."—*American Medico-Surgical Bulletin.*

"Nothing at once so scientific and so simple has appeared on the subject. It is unreservedly commended as a text-book for secondary schools."—*The Educational Review.*

GALTON.—Healthy Hospitals. Observations on some points connected with Hospital Construction. With Illustrations. By SIR DOUGLAS GALTON, K.C.B., F.R.S. $2.75.

"This is a valuable contribution to the literature of a most important subject. The eminence in engineering circles of Sir Douglas Galton would alone determine its merit. After a brief introduction into the origin of hospitals, he defines them as places 'not only for the reception and cure of the sick poor . . . but also as technical schools in which the medical student must learn his profession, and as experimental workshops in which the matured physician or surgeon carries on scientific research.' . . . While the subject-matter is largely technical, it is presented in a clear style, and its meaning is clear to any intelligent person. We commend Sir Douglas Galton's book to all builders of hospitals."—*Medical Record.*

GAMGEE.—A Text-book of the Physiological Chemistry of the Animal Body. Including an Account of the Chemical Changes occurring in Disease. By ARTHUR GAMGEE, M.D., F.R.S. 8vo.

Vol. I. The Proteids. $4.50.

Vol. II. The Physiological Chemistry of Digestion. With two chromo-lithographic Charts by Spillon and Wilkinson. $4.50.

GILLIES.—The Theory and Practice of Counter-irritation. By H. CAMERON GILLIES, M.D. 8vo. $2.50.

GOODFELLOW (J.).—**The Dietetic Value of Bread.** $1.50.

GRESWELL (D. A.).—**A Contribution to the Natural History of Scarlatina.** 8vo. $2.50.

GRIFFITHS (W. H.). — **Lessons on Prescriptions.** 18mo. $1.00.

HAMILTON (D. J.). — **A Systematic and Practical Text-book of Pathology.**

Vol. I. Technical. General Pathological Processes. Diseases of Special Organs. 8vo. $6.25.

Vol. II. Diseases of Special Organs (*continued*). **Bacteriology, etc.** 2 Parts. 8vo. Each $5.00.

"This is beyond question the most complete work on Pathology in the English language to-day. The author has accomplished his laborious task most successfully. We cannot better criticise it than by saying it is beyond criticism." — *Canada Medical Record.*

HAVILAND. — **The Geographical Distribution of Disease in Great Britain.** By ALFRED HAVILAND. Second Edition. 8vo. $4.50.

HAWKINS (H. P.). — **On Diseases of the Vermiform Appendix.** With a consideration of the symptoms and treatment of the resulting forms of Peritonitis. By HERBERT P. HAWKINS, M.A., M.D., F.R.C.P. 8vo. Cloth, pp. 139. $2.25, *net.*

HILTON (J.). — **Rest and Pain.** Edited by W. H. A. JACOBSON. $2.00.

HOBLYN. — **Dictionary of Medical Terms.** Twelfth Edition. $2.25.

HULLAH (J.). — **The Cultivation of the Speaking Voice.** *Clarendon Press Series.* Second Edition. 16mo. 60 cents.

HUXLEY (T. H.). — **Lessons in Elementary Physiology.** With numerous Illustrations. Revised by Dr. FOSTER. 16mo. $1.10.

"Unquestionably the clearest and most complete elementary treatise on this subject that we possess in any language. In this admirable little work, the outlines of the physiology of the human body are set forth in the plainest English, with a simplicity and earnestness of purpose which command our highest admiration." — *Westminster Review.*

JENNER (Sir WILLIAM). — **Lectures and Essays on Fevers and Diphtheria, 1849-1879.** 8vo. $4.00.

"This volume is a fitting exemplar of the careful and scientific work that has placed the author in the foremost rank of his profession. It cannot fail to prove interesting to physicians." — *New York Medical Journal.*

Clinical Lectures and Essays on Rickets, Tuberculosis, Abdominal Tumours, and Other Subjects. 8vo. $4.00.

JEX-BLAKE (SOPHIA). — **The Care of Infants.** 18mo. 40 cents.

KANTHACK (A. A.). — **A Course of Elementary Practical Bacteriology, including Bacteriological Analysis and Chemistry.** By A. A. KANTHACK, Lecturer on Pathology and Bacteriology, St. Bartholomew's Hospital, and J. H. DRYSDALE. 12mo. Cloth. $1.10.

KIMBER. — **Text-book of Anatomy and Physiology for Nurses.** Compiled by DIANA CLIFFORD KIMBER, graduate of Bellevue Training School; Assistant Superintendent New York City Training School, Blackwell's Island, N. Y.; formerly Assistant Superintendent Illinois Training School, Chicago, Ill. Fully Illustrated. 8vo. $2.50.

"A well written and unusually thorough work for its purpose. Although intended for nurses, it would make an admirable text-book in our schools and academies." — *American Medico-Surgical Bulletin.*

KLEIN (E.). — **Micro-organisms and Disease.** Revised, with 120 ings. $1.00.

The Bacteria in Asiatic Cholera. $1.25.

KOCHER. — **Text-Book of Operative Surgery.** By Dr. THEODOR I Professor of Surgery and Director of the Surgical Clinic in the Univ Bern. Translated with the Special Authority of the Author from the Revised and Enlarged German Edition by HAROLD J. STILES, M.B., I Edin., Senior Demonstrator of Surgery and formerly Demonstrator of A in the University of Edinburgh, etc. With 185 Illustrations. 8vo. $

MACALISTER. — **A Text-Book of Physical Anthropology.** *In*

MACDONALD (GREVILLE). — **A Treatise on Diseases of the No its Accessory Cavities.** Second Edition. 8vo. $2.50.

MACEWEN. — **Pyogenic Infective Diseases of the Brain and Cord, Meningitis, Abscess of Brain, Infective Sinus Thron** By WILLIAM MACEWEN, M.D., Glasgow. 8vo. Buckram. $6.00.

Atlas of Head Sections. 53 Engraved Copperplates of Frozen Sec the Head, and 53 Key Plates with Descriptive Tests. Folio. B buckram. $21.00.

"The 'Atlas' should certainly be in the hands of every surgeon who aspires to enter tl brain surgery, as a careful inspection of these plates will teach more than many volumes wri the subject. . . . The plates themselves have been executed with the greatest care, the ill being very fine photogravures from photographic plates, about two-thirds of the actual s head. The appearance of the book is a credit both to the author and to the publisher." — *Record.*

M'KENDRICK (J. G.). — **A Text-book of Physiology.** In 2 volum

Vol. I. General Physiology. 8vo. $4.00.

Vol. II. Special Physiology. 8vo. $6.00.

Life in Motion; or, Muscle and Nerve. Illustrated. $1.50.

MACLAGAN (T.). — **The Germ Theory.** 8vo. $3.00.

MACLEAN (W. C.). — **Diseases of Tropical Climates.** $3.00.

MARSHALL. — **Pain, Pleasure, and Æsthetics.** By HENRY RUTGEI SHALL. 8vo. $3.00.

"The book must necessarily have a deep interest for physicians, and especially neurol it is concerned largely with the explanation of a phenomenon — pain, the prevention and which form such a good portion of their duty. We can heartily recommend it to their pe study." — *Journal of Nervous and Mental Disease.*

MAUDSLEY. — **The Pathology of Mind: A Study of its Diste Deformities, and Disorders.** Second Edition. By HENRY MA M.D. 8vo. Cloth, pp. 571. $5.00, *net.*

MERCIER (C.). — **The Nervous System and the Mind.** 8vo. $4.0

MIERS (H. A.) and **CROSSKEY** (R.). — **The Soil in Relation to I** By HENRY A. MIERS and ROGER CROSSKEY. $1.10.

MIGULA (W.). — **An Introduction to Practical Bacteriology.** Tr by M. CAMPBELL, and edited by H. J. CAMPBELL, M.D. Illustrated.

"Its practical character fits it for a guide for students desiring a knowledge of the el principles of this interesting and important topic." — *Popular Science Monthly.*

MIVART (ST. GEORGE).—**Lessons in Elementary Anatomy.** With 400 Illustrations. 16mo. $1.75.

PALMBERG (A.).—**A Treatise on Public Health and its Application in Different European Countries** (England, France, Belgium, Germany, Austria, Sweden, and Finland). Edited by ARTHUR NEWSHOLME. 8vo. $5.00.

"The book is rich in descriptions and illustrations of sanitary appliances, modern and practical." —*Popular Science Monthly.*

PIFFARD (H. G.).—**An Elementary Treatise on Diseases of the Skin.** With Illustrations. 8vo. $4.00.

REYNOLDS.—**A Primer of Hygiene.** By ERNEST S. REYNOLDS, M.D. (Lond.), Senior Physician to the Ancoats Hospital, Manchester, etc., etc. With 50 Illustrations. 18mo. 35 cents.

"This is a rare bit of text-book making. The pedagogical features are eminently creditable; the literary work, from the text-book standpoint, is admirable. The illustrations are simple, but ideal in their clearness and illustrative qualities."—*Journal of Education.*

RICHARDSON.—Works by B. W. RICHARDSON, M.D.

On Alcohol. Paper, 30 cents.

Diseases of Modern Life. New Edition. (*In the Press.*)

ROLLESTON and **KANTHACK.**—**Manual of Practical Morbid Anatomy.** Being a Hand-book for the Post-mortem Room. *Cambridge Natural Science Manuals: Biological Series.* 12mo. $1.60.

"To those interested in post-mortem work, this book can be heartily recommended as a most valuable, complete, and efficient guide."—*American Medico-Surgical Bulletin.*

STEVEN (J. L.).—**Practical Pathology.** $1.75.

STRAHAN.—**Suicide and Insanity.** A Physiological and Sociological Study. By S. A. K. STRAHAN, M.D., Author of "Marriage and Disease," "Instinctive Criminality," etc. 12mo, cloth. $1.75.

"The subject treated of in Dr. Strahan's excellent little work has always possessed, and, in the nature of things, must in future possess keen interest, not only for physicians and jurists, moralists and sociologists, but for all thinking persons. That such a fundamental instinct as the desire to live should ever so far yield, either to reason or imperative craving, as to culminate in self-destruction, is a phenomenon so opposed to the principles of animal existence as to affect even the most unimaginative very much as would the sudden reversal of the law of gravitation."—*Medical Record.*

"The book is a most interesting contribution to the literature of a subject that is of increasing interest to the alienist and psychologist."—*N. Y. Medical Journal.*

"The author has presented a very interesting and unbiased study of a topic that is engaging more and more attention, for it is not one of the least of the charges against modern society that its organization is such that men and women are unwilling to continue as associates thereof."—*Popular Science Monthly.*

THORNE.—**Diphtheria.** Its Natural History and Prevention. By R. THORNE. $2.00.

VON KAHLDEN.—Methods of Pathological Histo
KAHLDEN, Assistant Professor of Pathology in the Uni
Translated and Edited by H. MORLEY FLETCHER, M.D., (
St. Bartholomew's Hospital, and Assistant Demonstrator
Medical School. With introduction by G. SIMS WOODHEA
the Laboratories of the Royal Colleges of Physicians and S

"This manual of methods recommends itself to every student of histol
form of footnotes are presented additions and modifications of great practica
book up to date. There are chapters on the microscope and apparatus requis
examination of fresh tissues, methods of hardening, decalcification of tissues
infection of tissues, preparing and mounting sections, staining processes,
animal parasites, and microscopical examinations for medico-legal purposes.'

WHITE (W. HALE, M.D.). — **A Text-book of General Th**

"In no other text-book of Therapeutics can the same interesting, varied
be found so usefully, successfully, and agreeably presented." — *N. E. Medi*

WILLIAMS. — Aero-Therapeutics. The Treatment of
Climate. Being the Lumleian Lectures for 1893 delive
College of Physicians. With an address on the high a
By CHARLES THEODORE WILLIAMS, M.D., Senior Physi
for Consumption and Diseases of the Chest, Brompton, a
the Royal Meteorological Society. 8vo, cloth. $2.00.

"It is not an exhaustive work, it is true, but it is sufficiently full for
accurate, practical, and charmingly written. For those who wish a small wo
ment of pulmonary diseases, written by a recognized authority on the subjec
cally and usefully most reliable information on a very important subject, we
Dr. Williams's work." — *N. Y. Medical Journal.*

WILLOUGHBY (E. F.). — **Handbook of Public Health**
16mo. $1.50.

"An admirably concise and lucid treatment of preventive medicine, alike
readers, teachers, students, physicians, and sanitary inspectors." — *The San*

ZIEGLER. — A Text-book of Pathological Anatomy a
With Illustrations. 8vo.

Part I. Special Pathological Anatomy. Sections I
Part II. Special Pathological Anatomy. Sections I
Part III. General Pathological Anatomy. *In prepa*

ZIEHEN (THEODORE). — **Introduction to Physiologi**
Translated by C. C. VAN LIEW and Dr. OTTO BEYER. V
$1.50.

"The book can be highly recommended to all who wish a short and clea
— *Educational Review.*

"The general reading of this book would, we think, do more for the sch
investment of the same amount of time and money in anything else." — *Jou*

THE MACMILLAN COMPA
NEW YORK: 66 FIFTH AVENU
CHICAGO: ROOM 23, AUDITORIUM.

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305

L41 Allbutt,T.C. 3854
A421 System of medicine
v.4
1898

NAME | DATE DUE